77

Environmental Assessment

Waste Treatment Facility

OWMC

Ontario
Waste Management
Corporation

A provincial crown corporation.

SITE SELECTION PROCESS
PHASE 4A:
SELECTION OF A PREFERRED SITE(S)

GENERIC RISK ASSESSMENT

Prepared for
The Ontario Waste Management Corporation

By

Environ Corporation
Washington, D.C. and Princeton, N.J.
U.S.A.

December 6, 1985

REVISED

ENVIRON

ENVIRON Corporation
Counsel in Health and Environmental Science

October 6, 1986

Ontario Waste Management Corporation
2 Bloor Street West
11th Floor
Toronto, Ontario
Canada H4W 3E2

Re: Generic Risk Assessment, Volumes 1 and 2

Dear Sirs:

We are pleased to submit our Generic Risk Assessment, Volumes 1 and 2. Volume 1 consists of the technical report, which has been modified to incorporate minor changes to the original report issued December 6, 1985. Volume 2 consists of appendices of supporting technical information. It should be noted that the quantitative estimates of public health risks provided in this report are not final. The generic risk assessment will be expanded in Phase 4B to incorporate refinements to the assumptions and methodology used to characterize human exposure.

Sincerely,

Joseph H. Highland, Ph.D.
Principal

LMT:slh

Enclosure

The Flour Mill, 1000 Potomac St., N.W.
Washington, D.C. 20007 • (202) 337-7444

VOLUME 1

Technical Report

CONTENTS

Page

EXECUTIVE SUMMARY i

1. INTRODUCTION 1
   1.1 Background and Purpose 1
   1.2 Scope of Risk Assessment 4
   1.3 Relationship to Other Site Selection Activities 7
   1.4 Organization of Report 8

2. THE RISK ASSESSMENT PROCESS 12
   2.1 Principles of Risk Assessment 12
   2.2 Limitations and Uncertainties of the Risk Assessment Process 20
   2.3 Application of the Risk Assessment Process to the OWMC Facility 21

3. CHARACTERIZATION OF RELEASES 34
   3.1 Characteristics of Facility 34
   3.2 Major Sources of Releases 36
   3.3 Selection of Surrogate Chemicals 41
   3.4 Estimation of Release Rates 45

4. DIRECT EXPOSURES TO FACILITY EMISSIONS 51
   4.1 Pathway Definition 51
   4.2 Long Term Exposures 52
   4.3 Short Term Exposures 55

5. INDIRECT EXPOSURES TO FACILITY EMISSIONS 63
   5.1 Pathway Definitions 63
   5.2 Ingestion of Leafy Vegetables 64
   5.3 Ingestion of Beef and Milk 69
   5.4 Ingestion of Cistern and Pond Water 70
   5.5 Exposure Assessment and Risk Characterization 72

6. EXPOSURE VIA TRANSPORTATION SPILLS 75
   6.1 Pathway Definition and Spill Frequency 75
   6.2 Exposure Assessment and Risk Characterization 79

7. CONCLUSIONS 83
   7.1 General 83
   7.2 Acute Exposures 84
   7.3 Chronic Exposures 86
   7.4 Ranking of Risk Factors 88

GLOSSARY 96

REFERENCES 103

LIST OF TABLES

		Page
Table 1	Risk Factors for Comparison of Siting Options	5
Table 2	Inputs to Risk Assessment From Other Consultant Studies	9
Table 3	Assumptions Used In Exposure Assessment	23
Table 4	Standard Assumptions for Human Dose Calculations	26
Table 5	Sources of Environmental Releases	38
Table 6	Selection of Surrogate Chemicals	43
Table 7	Dispersion Factors for Long Term Emissions	53
Table 8	Dispersion Factors for Short Term Emissions	56
Table 9	Risk Estimates for Acute Exposures from Facility Upsets	57
Table 10	Estimates of Lifetime Cancer Risk for Chronic Exposure from Indirect Pathways	74
Table 11	Frequency of Truck Accident-Related Spills of Special Wastes on Local and Regional Access Roads	78
Table 12	Risk Estimates for Acute Exposures	84
Table 13	Estimates of Lifetime Cancer Risk for Chronic Exposures	87
Table 14	Risks of Death from Voluntary and Involuntary Exposures	89

LIST OF FIGURES

Figure 1	Facilities Development Process 4A	2

EXECUTIVE SUMMARY

PURPOSE OF THE REPORT

The Ontario Waste Management Corporation (OWMC) is engaged in a Facilities Development Process to establish a hazardous waste management facility for the Province of Ontario. OWMC's most highly ranked goal in the siting of the facility is the minimizing of risk to human health. A generic risk assessment was undertaken, therefore, as part of the OWMC siting process, in order to provide OWMC with a better understanding of the relative importance of siting factors related to human health risk. The generic risk assessment was designed to provide quantitative estimates of public health risks associated with emissions of toxic pollutants from the proposed hazardous waste treatment and disposal facility . The term "generic" indicates that at this stage the risk assessment has used a single set of generalized site parameters applicable to all sites rather than characteristics specific to each candidate site.

Volume 2 of this report includes appendices of supporting technical information.

SCOPE AND METHOD

In performing the risk assessment, consideration was given to both long-term exposures associated with normal facility operations, as well as short-term exposures associated with facility upset conditions or accidents, and off-site transportation accidents. For the long-term exposures, consideration was given not only to direct inhalation of air pollutants, but also to exposure via drinking water from ponds and cisterns; consumption of crops; and consumption of beef and milk from local livestock. Only direct inhalation was considered for the short-term exposures.

For each of these risk pathways, human exposure was assessed in a four step process which can be summarized as: (1) identification of significant sources of environmental release; (2) characterization of the selected releases in terms of the nature of the chemicals, frequency and duration of release, and magnitude of release; (3) modeling of pollutant transport in the environment; and (4) estimation of human dose by inhalation or ingestion.

Health risk was characterized in terms of both acute and chronic effects with consideration of exposure to both carcinogenic and non-carcinogenic chemicals. In the case of carcinogens, cancer risk values were calculated and compared to a lifetime cancer risk of one additional cancer per million people exposed. This value was used as an acceptable level of risk. For non-carcinogens, the estimated human dose based on the predicted exposures was compared to a toxicologically safe level. The ratio of the safe level to the predicted exposure level was termed the adjusted margin of safety or AMOS value.

PREMISES AND POSTULATES

The risk assessment was undertaken for a proposed facility capacity of 300 000 tonnes per annum of special wastes. The details of the proposed facility and characteristics of the wastes to be accepted at the facility were provided by OWMC. Risk estimates were based on the preliminary design of the facility and did not include any provision for mitigation of potentially adverse health effects.

Throughout the risk assessment a series of conservative assumptions was made in defining the scenarios for the characterization of facility releases, environmental transport modeling and estimation of human dose. These assumptions were considered conservative in that they resulted in the selection of facility operating parameters and environmental conditions that would tend to overestimate the magnitude of human exposures experienced. As a result of these conservative assumptions, the predicted human health risks are likely overstated.

STATUS OF DATA

Data used in the risk assessment were provided from two principal sources: published literature on the toxicological behavior of chemicals in the environment -- their transport, fate and effect; and other consultant studies undertaken for OWMC. Two key data elements can be identified in the risk assessment process: the types and quantities of chemicals emitted; and the effects of these chemicals on human health.

For characterizing emissions of potentially toxic pollutants, the risk assessment was based on the preliminary design of the facility and on available data on waste characteristics. While the design of the facility will be refined in subsequent stages of OWMC's Facilities Development Process, the existing design information was considered sufficiently detailed to quantify the magnitude and duration of emissions. Information on the frequency of facility upset conditions and accidents, however, was limited to qualitative estimates only.

Data on waste types to be managed by the proposed facility were based on a survey of 1000 industries in Ontario. A limited amount of quantitative data on waste constituents was available from this source. To account for lack of full information, chemical constituents of concern in individual waste streams were identified, and the most hazardous chemicals used to represent particular waste categories in a way which would tend to overestimate risk. It was not yet possible, however, to provide data on landfill leachate characteristics in a form which could be employed in a quantitative risk assessment.

In the case of the toxicological effects of the chemicals on human health, the risk assessment relied on a mixture of human experience and animal experiments. While there are limitations and uncertainties in extrapolating from such data, the use of appropriate safety factors and mathematical models is an accepted approach that provides a systematic approach to evaluating scientifically the available data.

Overall, it was considered that the use of conservative assumptions and safety factors enabled meaningful and useful estimates of risk to be derived for the risk pathways addressed.

MAIN FINDINGS

In the case of short term exposures occurring as a result of episodic release events, the risk assessment was based on the potential for acute effects. Acute effects were considered to include very mild effects such as odor or minor irritation, although these should not necessarily be considered adverse to health. The potential for acute effects was considered significant when the AMOS value (defined as the ratio of a toxicologically safe level to the predicted exposure level) was less than one. The AMOS value was less than one in three cases: the incinerator stack scrubber failure, transportation spill and tank farm fire. In the case of the incinerator stack scrubber failure, further evaluation of the toxicological literature indicated that no adverse health effects would be expected, although slight odor or minor, reversible irritation effects might be experienced. For the transportation spill and tank farm fire, further evaluation indicated that potentially severe health effects could be experienced. Both events, however, are believed to have low probabilities of occurrence, and it should be emphasized that the severity of the actual adverse health effect would be strongly influenced by such factors as distance of the human receptor from the spill or fire, the duration of exposure and the amount of toxic pollutant released. Also, it should be noted for the transportation spill that only 8% of the special wastes transported to the facility have the potential to cause effects under the spill conditions considered here.

In the case of chronic exposures, health risk was evaluated based on the magnitude of potential cancer risk associated with each exposure pathway. In all cases, the predicted cancer risks were equal to or less than one in a million (1×10^{-6}).

CONCLUSIONS

The risk estimates developed in the generic risk assessment are considered to overstate the incremental risk associated with OWMC's hazardous waste management facility. Although these risk estimates are appropriate for site selection purposes, they should not be considered as measures of absolute risk.

For short term events, the potential for adverse health effects was predicted only for the transportation spill and tank farm fire. The risks associated with these events were considered to be comparable and more significant than those for the incinerator scrubber failure.

For chronic emissions, the long term health risks, both individually and in combination, were all judged acceptable relative to the one in a million cancer risk criterion established for this study. The risks from direct inhalation of both fugitive and stack emissions, however, were more significant than those from the indirect pathways of drinking of water from ponds and cisterns, consumption of contaminated crops, or consumption of contaminated meat and milk.

RECOMMENDATIONS

The health risk assessment provides quantitative data upon which comparisons of risk pathways can be made. It is recommended that these comparisons of risk pathways be used by OWMC to assist in the ranking of risk factors as input to the selection of a preferred site(s) for the proposed facility.

Further, it is recommended that a more detailed, site-specific risk assessment be undertaken for the selected site. To facilitate this more detailed risk assessment, consideration should be given to improving the waste characteristics data base, both for the wastes to be received by the facility and for the leaching characteristics of the solidified wastes to be disposed of in the engineered landfill. Also, a hazard analysis of the facility should be undertaken to provide more detailed information on potential accidents and upset conditions, and, in particular, on their frequency of occurrence.

CHAPTER 1

INTRODUCTION

1.1 BACKGROUND AND PURPOSE

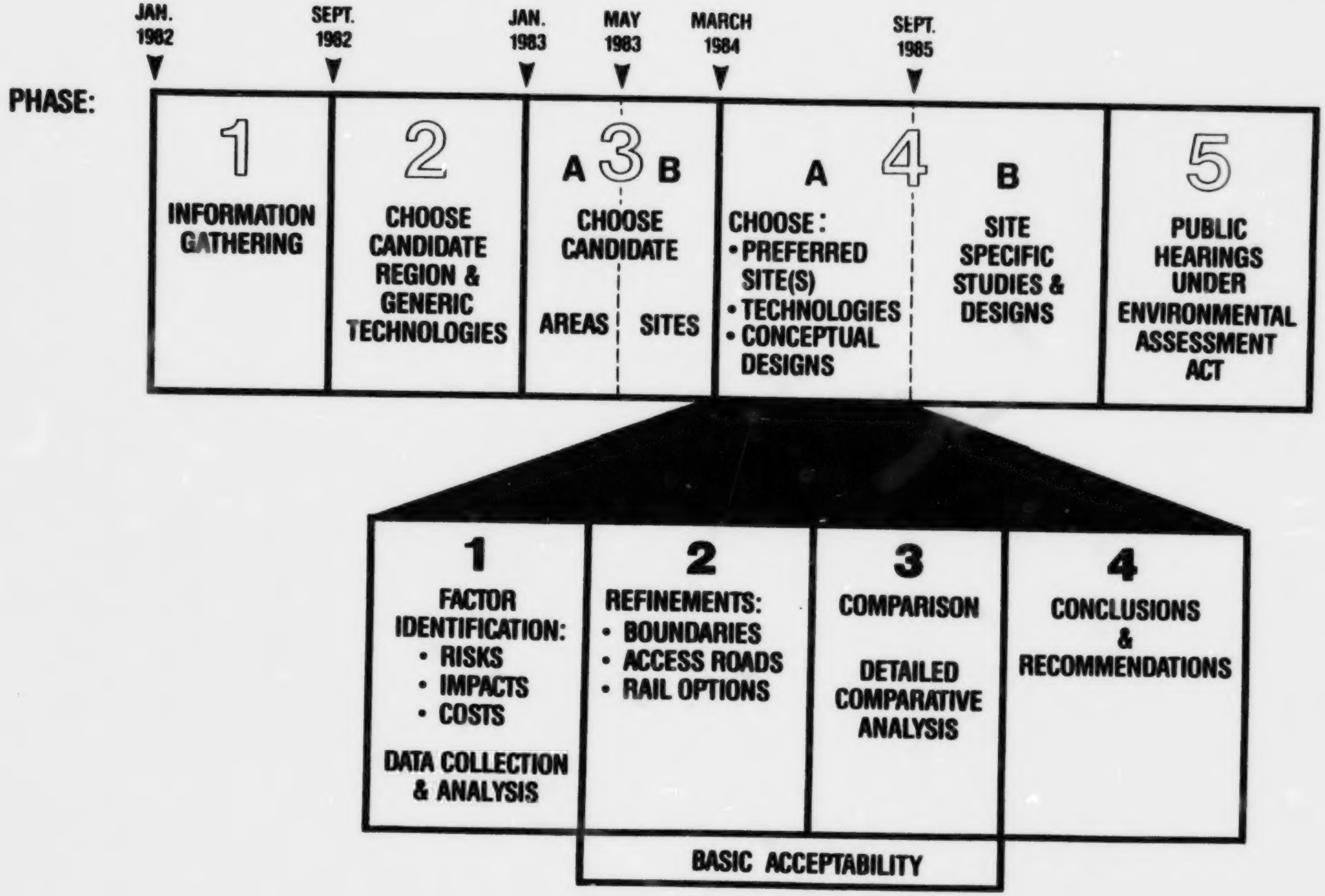
The Ontario Waste Management Corporation (OWMC) is engaged in a Facilities Development Process to establish a hazardous waste management facility for the Province of Ontario (OWMC, 1985). The Facilities Development Process consists of five phases designed to move OWMC through the process of selecting a site(s) and designing the facilities in preparation for the hearings and approval process required prior to construction (see Figure 1). Throughout this process the primary goal of OWMC has been and continues to be the minimizing of risk to public health.

A major part of the Facilities Development Process is the selection of a preferred site(s) for the proposed hazardous waste management facility. This site selection process has involved a series of successively more detailed steps to identify potentially suitable sites. At the end of Phase 3 of the Facilities Development Process, eight candidate sites were identified located in the Niagara Falls, West Lincoln, Mississauga and Milton areas.

To select a preferred site(s), Phase 4A of the Facilities Development Process consists of a comparative evaluation of the eight candidate sites (M.M. Dillon, 1985a). Three major study components are addressed within

FIGURE 1

FACILITIES DEVELOPMENT PROCESS 4A


JAN. 1982
SEPT. 1982
JAN. 1983
MAY 1983
MARCH 1984
SEPT. 1985
PHASE:
1
INFORMATION GATHERING
2
CHOOSE CANDIDATE REGION & GENERIC TECHNOLOGIES
A 3 B
CHOOSE CANDIDATE
AREAS
SITES
A 4 B
CHOOSE:
• PREFERRED SITE(S)
• TECHNOLOGIES
• CONCEPTUAL DESIGNS
SITE SPECIFIC STUDIES & DESIGNS
5
PUBLIC HEARINGS UNDER ENVIRONMENTAL ASSESSMENT ACT
1
FACTOR IDENTIFICATION:
• RISKS
• IMPACTS
• COSTS
DATA COLLECTION & ANALYSIS
2
REFINEMENTS:
• BOUNDARIES
• ACCESS ROADS
• RAIL OPTIONS
3
COMPARISON
DETAILED COMPARATIVE ANALYSIS
4
CONCLUSIONS & RECOMMENDATIONS
BASIC ACCEPTABILITY

this comparative evaluation - Risks, Impacts and Costs. This summary report is concerned with those issues related to human health risk associated with exposure to toxic substances.[1] Within this category, the site selection process identifies the following risk factor groupings with regard to the potential effects of a waste management facility (M.M. Dillon, 1985a):

- Transportation Risks
- Air Dispersion/Air Quality Risks
- Risks Related to Hydrogeologic Suitability
- Risks Related to Surface Water Suitability

Risks in these factor groupings are associated with potential releases of chemicals during the normal operation of the facility, such as fugitive and stack emissions; with releases that might occur during facility upsets or accidents; and with releases caused by transportation spills. These releases can result in contamination of air, surface water, groundwater and soil. Human exposure to the released contaminants can occur through inhalation, ingestion, or by skin absorption. A number of specific risk pathways (e.g., direct inhalation of contaminated air or ingestion of contaminated crops) have been selected for analysis. Once the risk associated with each pathway is understood, a comparison can be made to assist in the weighting of siting factors related to human health. The purpose of the generic risk assessment is to provide the data on which such comparisons can be made. The term "generic" indicates that at this stage the risk assessment has used a single set of generalized parameters applicable to all sites rather than characteristics specific to each candidate site. A site-specific risk assessment should be undertaken as part of the environmental impact assessment of the preferred site.

---

[1] Appendices of supporting technical information are presented in Volume 2 of this report.

1.2 SCOPE OF RISK ASSESSMENT

The risk assessment process was designed to provide estimates of human health risk associated with exposure to toxic pollutants via selected risk pathways. The pathways selected for quantitative analysis were chosen from the risk factors identified by OWMC for comparison of siting options. These risk factors are summarized in Table 1, and discussed further in other Phase 4A reports (M.M. Dillon, 1985a; OWMC, 1985). The selected pathways were: (1) those which were believed to have the potential to contribute most significantly to public health risk; (2) those which would address specific expressed public concerns; and (3) those for which data were in a form suitable for quantitative risk assessment. The pathways selected consisted of the following:

1. Exposure resulting from normal facility operations:
    a. Direct inhalation of air pollutants,
    b. Ingestion of contaminated drinking water from ponds and cisterns,
    c. Consumption of contaminated crops,
    d. Consumption of contaminated meat from livestock which ingested chemical contaminants in their food and water,

TABLE 1

RISK FACTORS FOR COMPARISON OF SITING OPTIONS

TRANSPORTATION

- Annual province-wide hazard-based spill index of OWMC generated transport
- Annual province-wide accident involvement of OWMC generated transport
- Transportation risks on local and regional access roads due to accident involvements of OWMC generated transport
- Transportation risks on local and regional access roads due to hazard-based spills of OWMC generated transport

---

AIR DISPERSION/AIR QUALITY

- Risks from direct inhalation of emissions - chronic
- Risks from direct inhalation of emissions - acute
- Risks from consumption of cistern and pond water
- Risks from consumption of crops and livestock

HYDROGEOLOGIC SUITABILITY

- Capability of providing natural containment
- Ground water use

SURFACE WATER SUITABILITY

- Susceptiblity of site to flooding
- Potential for water use interference

Source: M.M. Dillon, 1985a.

   e. Consumption of contaminated milk from livestock which ingested chemical contaminants in their food and water.

2. Exposures resulting from facility upset conditions.
3. Exposures resulting from facility accidents.
4. Exposures resulting from transportation accidents.

Each of the selected pathways noted above is associated with specific air emissions from the facility or from a transportation accident. Releases to groundwater and the potential for exposure initially were to be considered as well. However, the groundwater pathway was not considered because sufficiently detailed data on the potential leachate quality from the landfill and on site-specific hydrogeology were not available at this time. Information on the site-specific hydrogeology will be available at a later stage in OWMC's Facilities Development Process, when detailed testing of the preferred site(s) has been undertaken. In the case of the leachate quality, it is recommended that the leaching characteristics of the solidified wastes to be deposited in the landfill be investigated further. In the meantime, the focus of the groundwater pathway analysis has been on a qualitative comparison of siting options as described by Gartner Lee Associates (1985).

The exposures considered potentially significant as a result of normal facility operations were both fugitive emissions from the handling of materials at the facility and incinerator stack emissions from the combustion of waste material. Each of these can contribute to a direct impact on air quality and thus were considered significant in evaluating the potential for the direct inhalation of chemical contaminants. It should be noted that the point of maximum impact created by fugitive and stack emissions occurs at different locations, and that the health risk assessment considered the highest potential for exposure in each case. This approach, the use of conservative assumptions, is consistent with the approach used throughout the risk assessment process.

In addition to creating direct impacts on air quality and thus potential human exposures, both fugitive and stack emissions can lead to indirect human exposures. As part of the risk assessment, the potential ingestion of contaminated drinking water from ponds and cisterns was considered. In a similar manner, the potential for ingestion of contaminated crops was considered, and the ingestion of contaminated meat and milk from livestock raised on contaminated crops was evaluated.

Consideration was given also to exposure resulting from facility upset conditions and accidents, and transportation accidents. Each of these events can result in emissions of volatile organic chemicals. Direct exposures only were considered for these emissions, and health risk was assessed at the point of maximum exposure in each case.

1.3 RELATIONSHIP TO OTHER SITE SELECTION ACTIVITIES

The generic risk assessment forms part of a series of studies undertaken by a team of consultants. The risk assessment is linked to these other site selection activities in that the bases for many of the inputs to the risk assessment were provided by other consultant studies. For example, the sources of potential releases were identified by review of the proposed facility design (Monenco, 1985a) and surrogate chemicals for modeling these releases were selected by an analysis of a data base on waste types expected to be received at the facility. The development of the database is described in a report prepared for OWMC by Canviro Consultants, Ltd. (1985). Other inputs to the risk assessment were provided directly by other consultants, e.g. the air dispersion modeling of chemical releases and the estimation of the deposition rates for these chemicals were conducted by the Ontario Research Foundation. These inputs to the risk assessment process and how they were used are summarized in Table 2.

The overall purpose of the risk assessment was to assist in the weighting of siting factors related to human health. To accomplish this, it drew on data from studies by other consultants and in turn generated risk estimates for use by these other consultants. These risk estimates were used as part of the overall ranking of risk factors (M.M. Dillon, 1985a) and particularly in the consideration of atmospheric dispersion (ORF, 1985b).

1.4 ORGANIZATION OF REPORT

This report is organized into seven chapters.

- Chapter 1, Introduction (this chapter), provides an overall description of the risk assessment process and why it was undertaken.

TABLE 2

INPUTS TO RISK ASSESSMENT FROM OTHER CONSULTANT STUDIES

Input Data	Source	Analysis undertaken in Risk Assessment
Facility design	Monenco	Identification of potential release sources
Waste stream characteristics	Canviro	Selection of surrogate chemicals
On-site gas emissions and calculations	Monenco	Estimation of chemical-specific emissions
Empirical emissions data	ORF	Estimation of chemical-specific emissions
Definition of facility upset and accident conditions	M.M. Dillon Monenco	Estimation of chemical-specific emissions
Transportation spill frequency	M.M. Dillon	Estimation of exposure probability for spill events
Air dispersion modeling	ORF	Estimation of chemical concentrations at exposure points
Chemical deposition rates	ORF	Estimation of chemical concentrations in cisterns and ponds.
		Estimation of chemical concentrations in soil and vegetables.
Livestock and crop production data	Ecologistics	Estimation of chemical concentrations in vegetables, meat and milk

- Chapter 2, The Risk Assessment Process, provides a description of the principles of risk assessment and discusses the limitations inherent in any risk assessment. Also included is a discussion of the application of risk assessment to the OWMC facility siting process.

- Chapter 3, Characterization of Releases, contains a description of the characteristics of the facility and the major sources from which releases may occur. Each release subsequently is evaluated based on the use of surrogate chemicals.

- Chapter 4, Direct Exposures to Facility Emissions, presents the analyses of the risk pathways involving direct inhalation of fugitive and incinerator stack emissions as well as those resulting from the direct inhalation of air emissions from facility upsets and accidents. In each case the risk pathway is defined, an assessment of human exposure is provided, and human health risk is characterized.

- Chapter 5, Indirect Exposures to Facility Emissions, presents an analysis of health risk associated with the ingestion of contaminated water from cisterns and ponds, and the consumption of contaminated food from either crops, meat or milk. The pathways are defined, human exposure is evaluated, and health risk is characterized.

- Chapter 6, Exposure via Transportation Spills, presents an analysis of the health risk associated with the inhalation of volatile organic compounds released in transportation spills. The pathway is defined, an estimate of spill frequency is developed, human exposure is evaluated, and health risk is characterized.

- Chapter 7, Conclusions, presents a summary of the findings of the risk assessment, and an analysis of the relative importance of these risks to each other and other common risks.

The description of the risk assessment process requires the use of certain terminology and technical language. Clarification of this terminology and technical language is provided in the report where appropriate, and a glossary of all technical terms follows the Conclusions of the report.

CHAPTER 2

THE RISK ASSESSMENT PROCESS

2.1 PRINCIPLES OF RISK ASSESSMENT

For the purposes of this study, human health risk can be defined as the probability of injury, disease, or death as a result of exposure to toxic substances. It can be expressed in numerical terms, and takes values from zero (absolute certainty that harm will not occur) to one (absolute certainty that it will occur). For some types of chemical exposures, risks can be estimated from historical data on the relationship between an activity and the incidence of adverse outcomes. For example, extensive documentation may exist on deaths and other types of injuries resulting from accidental poisoning by household products, pesticides, and therapeutic agents. These types of chemical exposures of short duration are termed acute exposures, and generally are associated with immediately observable effects. These effects may cease when the exposure ceases, i.e., acute effects, or they may persist beyond the period of exposure in which case the effects would be termed chronic.

Other types of chemical exposures do not produce immediately observable effects and it is only over a relatively long period of exposure, i.e., a chronic exposure, that adverse health effects may result. These adverse health effects are typically chronic effects, and generally are evaluated from animal studies.

Exposures associated with an operating hazardous waste management facility may be both acute, e.g., accidental emissions of relatively short duration and chronic, e.g., continuous emissions from fugitive sources. It is the purpose of this chapter to describe the process used to evaluate human health risks from such exposures.

In general, the risk assessment process consists of the following components:

- exposure assessment
- hazard evaluation
- risk characterization.

EXPOSURE ASSESSMENT

Knowledge of the routes, magnitude and duration of exposure to environmental chemicals is the first component of risk assessment. Human exposure is assessed typically in terms of the dose of chemical taken into the body, commonly in units of milligrams per kilogram of body weight per day. For certain gaseous pollutants, particularly those which act as irritants, exposure may be assessed in terms of the concentration to which an individual is exposed, commonly in milligrams per cubic metre or parts per million, and the time of exposure.

In the context of hazardous waste facilities, the starting point for assessing exposure is identifying the significant environmental releases associated with the waste management practices employed. For example, incineration may be characterized by emissions of incompletely combusted chemicals or hazardous combustion products, while physical/chemical treatment may be characterized by spills and fugitive emissions.

The next step is to identify possible routes of human exposure. Three routes -- inhalation, absorption and ingestion -- exist via which humans can become exposed to pollutants in their environment. These pollutants can be present in various environmental media such as air, surface water, groundwater, and soil. Through contact with these media, individuals can ingest, inhale or absorb pollutants and thus become exposed. If the concentrations of pollutants in each of these media are known or can be predicted and the extent and frequency of human contact with each is known, then human exposure to each pollutant can be estimated. To this end, environmental fate and transport models can be used to predict the concentrations of pollutants in the various environmental media likely to result from the operation of a hazardous waste management facility. To calculate the human dose that would result from contact with these pollutants, certain standard conventions are commonly applied. These conventions assume average values for the intake of food, water, etc., and also make assumptions regarding the absorption of pollutants within the human body. Estimates of human dose or pollutant concentration and exposure time calculated in this way can be applied to both short-term and long-term exposure assessments.

HAZARD EVALUATION

All chemical substances, whether natural or man-made, have the potential to cause some form of biological injury, disease or death under certain conditions of exposure. The term used to describe this inherent potential is toxicity, and the purpose of the hazard evaluation phase of risk assessment is to collect and evaluate information on the inherent toxic properties of the chemicals of interest. This information may relate both to acute and chronic toxic effects. Such information can be collected from two principal sources: epidemiological or clinical investigations, and experimental studies involving laboratory animals or other biological systems.

In addition to identifying the types of toxicity associated with a substance, it is also necessary to identify dose-response relationships. The frequency with which toxic effects appear in an exposed population (i.e., the risk) increases with increasing exposure or dose. In many cases, the types of toxic effects change as exposure increases, becoming more severe with increasing exposure and conversely, less severe with decreasing exposure. Thus, it is important to understand how the nature and magnitude of risk relate to any given dose.

A major dose-response issue is that of thresholds. A threshold dose is that which must be exceeded before a toxic effect is observed. The appearance of most toxic effects requires that a threshold be exceeded, although for some -- notably carcinogenic effects -- there appears to be a basis for rejecting the threshold hypothesis. In fact, carcinogenic risk assessment, as currently practiced, generally assumes the absence of thresholds. It should be noted, however, that this premise may not hold for all carcinogens (NRC, 1980).

One of the critical uses to which dose-response data are put is the identification of the dose at which no adverse response is observed in a population exposed to a toxic substance. This dose (called the "no-observed adverse effect level," NOAEL) may approximate a threshold dose for the population under study, and is generally taken as the starting point for risk assessment for chemicals that are assumed to operate by a threshold mechanism (NRC, 1980; Food Safety Council, 1980). This general approach applies to both acute and chronic exposures, although the dose-response data may be determined from different types of studies. Data for chronic exposures are generally derived from animal studies, although in some cases, particularly for exposure by inhalation, human data may be available from occupational settings. For acute exposures, human data are usually more available from clinical tests or accidental exposures, supplemented as necessary by information from animal studies.

In reviewing the dose-response data, it should be realized that not all measurable effects caused by chemical exposures are adverse to health, although for chronic exposures the no-observed-adverse-effect-level (NOAEL) typically is considered equivalent to a no-observed-effect-level (NOEL). For acute exposures, however, individuals may detect odors or experience certain minor forms of irritation which are reversible and will cease after exposure. These effects usually occur at low exposure levels long before adverse health effects are experienced. Thus, in evaluating the hazards for acute toxicants, a distinction can be made between the threshold for the effects that can impact human health (indicated by the

NOAEL) and the threshold for those effects that are expected to occur without adverse health impacts (indicated by the NOEL).

For carcinogens, the dose-response data are usually treated differently. To predict low dose risk from high dose/high risk data, which is the usual situation, it is necessary to apply certain mathematical models to the dose-response data (Food Safety Council, 1980; Klaasen, D.J. and Doull, J., 1980; IRLG, 1979). These models allow prediction of low dose risks, and their application yields an estimate of cancer risk per unit of dose (e.g., incidence of cancer at a dose of 1 mg/kg body weight/day, for continuous lifetime exposure). The hazard evaluation phase of risk assessment thus results in a presentation of NOAELs and/or NOELs for non-carcinogens, and of estimates of risk per unit dose (unit risks) for carcinogens.

RISK CHARACTERIZATION

Having evaluated the inherent hazard posed by a chemical agent, and the routes, magnitude and duration of the exposure, a quantitative measure of risk may be developed. A distinction is generally made here between the way in which risk is characterized for (1) chronic exposure to carcinogens; (2) chronic exposure to non-carcinogens; and (3) acute exposures.

For chronic exposures to non-carcinogens, it has become the practice to divide the experimentally-determined NOAEL by a large "safety factor" to estimate acceptable human doses. The application of the safety factor is based on the notion that there is likely to be a much wider range of susceptibilities in the human population than there is in the test animal group from which the NOAEL is obtained. The choice of safety factors depends on the nature and uncertainties of the toxicity test data. If the NOAEL was derived from a well-conducted animal chronic toxicity study, and has been carefully identified, the usual safety factor for estimating an "acceptable daily intake" (ADI) for humans is 100 (Food Safety Council, 1980; Federal Food, Drug and Cosmetic Act). Larger safety factors are commonly introduced when experimental data are lacking or limited, or when the effects of exposure are especially serious (Calabrese, 1983).

Risk characterization for non-carcinogens thus takes the form of determining the margin-of-safety (MOS) -- the numerical value derived when the experimental NOAEL is divided by the human dose. Under this system, the smaller the value of the MOS, the larger the risk. If there is virtually no MOS, it might be concluded that many members of the exposed population are at high risk of experiencing the form of toxicity experienced by the test animals at doses above the NOAEL. At a small fraction of the NOAEL, it is likely that the risk is very low and may be zero, although there is no practical way to determine whether this is actually the case.

For chronic exposures to carcinogens, the risk is characterized as the unit cancer risk. The risk per unit dose is multiplied by the actual daily lifetime dose experienced by people to derive an estimate of risk. In this case, an explicit risk is estimated, which takes values between zero and one. The definition of acceptable risk, e.g., 10^{-5}, 10^{-6}, etc., then becomes a matter of judgement or policy rather than a scientific determination. The use of numerical estimates of risk derived in this manner as decision-making tools for separating significant risk from insignificant risk has become common practice (NRC, 1980a; Rodricks, 1984; Rodricks and Taylor, 1983).

For acute exposures, criteria for short-term exposure limits for air pollutants have been developed by the National Academy of Sciences (NAS) (1979). NAS defines short-term exposure as a single exposure, usually lasting 60 minutes or less, but never more than 24 hours. The approach used depends on a substance-by-substance evaluation of each chemical. For compounds for which reliable human and animal data predict little chance of irreversible effects from a single exposure, exposure limits can be established directly from the human data with no additional safety factor. If the human data are weak but the animal data strongly suggest irreversible effects are unlikely from a single exposure, then a safety factor can be applied depending on the quality of the data. When cancer or some other irreversible effects are expected, then several possible approaches can be used. These include:

- Establish an exposure limit for the general population based on an appropriate safety factor as would be done for noncarcinogens; or, depending on the nature of the chemical, include specific warnings to particular segments of the population that may be at additional risk, e.g., in the case of exposure of pregnant women to a possible teratogen; or

- For carcinogens, use quantitative risk assessment to recommend an exposure level that provides a defined level of risk (i.e., 10^{-5}, 10^{-6}, etc.).

The appropriate choice depends upon the observed toxicological behavior of the chemical under review.

2.2 LIMITATIONS AND UNCERTAINTIES OF THE RISK ASSESSMENT PROCESS

Associated with the risk assessment framework are a set of uncertainties which can be considered in two categories: (1) those that are an inherent part of the current state of knowledge and cannot be improved upon; and (2) those that result from data deficiencies and can be improved upon by increased levels of sophistication or data input. Uncertainties of the first type are principally associated with hazard evaluation and include:

- Extrapolation of toxicity data from animals to humans
- Extrapolation of high dose/high risk data to low dose cases
- Lack of data on biological interaction and enhancement of toxic effects from combined exposures associated with hazardous materials.

Uncertainties of the second type apply more to the exposure assessment than to hazard evaluation. They can be improved upon to some extent by increased data collection (e.g., the development of additional waste characteristics data, the use of measured rather than estimated values for emissions or environmental concentrations), by the use of more sophisticated environmental fate and transport models, or by more detailed data on environmental conditions. Alternatively, where available data are limited, uncertainties can be handled, like the toxicological data limitations, by the use of safety factors or conservative assumptions.

While these limitations and uncertainties should be noted in the interpretation of the results of any risk assessment, the risk assessment methodology provides a systematic approach to evaluating scientifically the available data for assessing public health risk. With the use of appropriate assumptions and data inputs, risk assessment can provide a meaningful and useful measure of the potential for adverse health effects as a result of exposure to toxic substances.

2.3 APPLICATION OF THE RISK ASSESSMENT PROCESS TO THE OWMC FACILITY

The application of the risk assessment process to the OWMC facility is described in the following section in terms of the three components outlined previously: exposure assessment, hazard evaluation and risk characterization.

EXPOSURE ASSESSMENT

The exposure assessment consisted of the following four major steps:

(1) Identification of significant sources of environmental releases;

(2) Characterization of releases in terms of

- frequency and duration,
- chemicals to be modeled, and
- magnitude of release;

(3) Application of transport models to predict environmental concentrations;

(4) Estimation of human dose by inhalation or ingestion.

Throughout these steps in the exposure assessment, a series of conservative assumptions was made. These assumptions were considered conservative in that they resulted in the selection of facility operating parameters and environmental conditions that would tend to overestimate the magnitude of human exposures experienced. For example, data on waste stream characteristics were used to select the most hazardous waste stream within a class of wastes, and that class was modeled as if it were composed entirely of the most hazardous stream. Similarly, human exposure was estimated at the point of maximum exposure for all risk pathways considered. These and other conservative assumptions used in the exposure assessment are summarized in Table 3. Based on the use of such assumptions, the exposure estimate can be considered to overestimate actual exposure for an individual living or working in the vicinity of the facility.

The first step of the exposure assessment was to identify the significant sources of releases from the facility. The various operating units planned for the facility were reviewed by the engineering consultants (Monenco, 1985b), and a series of releases identified for further evaluation.

Second, the selected releases were characterized in terms of frequency and duration, the chemicals emitted and the magnitude of the

TABLE 3

ASSUMPTIONS USED IN EXPOSURE ASSESSMENT

CHARACTERIZATION OF RELEASES

- Waste classes represented by waste streams of highest hazard
- Waste streams represented by constituent of highest hazard
- Releases based on 300 000 t/a facility capacity
- Incinerator destruction and removal efficiency (DRE) of 99.99%
- Fugitive emissions based on all waste handled by each unit operation
- Transportation spills based on traffic accident involving loss of entire contents of truck
- Tank farm fire based on "boiling off" entire contents of tank
- No mitigation measures included beyond normal practice

POLLUTANT TRANSPORT MODELING

- Atmospheric conditions typical of Niagara region adopted for long term air dispersion modeling
- F stability conditions (worst-case) adopted for short term air dispersion modeling
- Scrubber failure emissions modeled as a truncated plume
- Plant uptake of all chemicals from soil based on highest partition coefficient recorded in literature surveyed

---

DOSE ESTIMATION

- All dose estimates based on exposure of off-site populations at point of maximum concentration
- Long term direct exposures based on continuous exposure over 30 year operating period
- Long term direct exposures to non-carcinogenic chemicals based on maximum two-week concentrations.
- Indirect exposures based on consumption of leafy vegetables, livestock products and water taken from point of maximum exposure
- Short term direct exposures from facility upset conditions and accidents based on exposure for duration of release
- Short term exposures from transportation spills based on exposure period of one hour

---

emission. The magnitude of the emissions was based conservatively on the maximum capacity of the facility, 300 000 t/a. Consideration was given to both long term releases under normal operating conditions as well as short term releases generated by upset conditions and accidents (both on-site and off-site).

Chemicals for each of these release types were chosen using a degree of hazard approach, whereby each category of waste was represented by the waste stream of highest hazard, and each waste stream represented by the chemical constituent of highest hazard. The approach accounts for the total quantity of wastes by assuming that the quantity of the highest hazard waste stream is equal to the quantity of the entire waste category. The degree of hazard approach was based on the toxicity, volatility and relative amount of each chemical expected at the facility, and was considered a conservative means to allow for some of the deficiencies in the data base for waste streams to be accepted by the facility. Emission rates for the selected chemicals were then estimated by a combination of empirical observations from comparable facilities (ORF, 1985a), and by engineering calculations (Monenco, 1985b).

The third step in the exposure assessment was the selection and application of appropriate models to predict multimedia environmental concentrations for the selected pathways, i.e., for direct exposures from inhalation and (for the long term releases) indirect exposures through consumption of local leafy vegetable crops, pond and cistern water, beef and milk.

In each case, conservative assumptions were used in the exposure assessment methodology: for long term releases, exposures were based on continuous exposure at the point of maximum concentration for a 30-year operating period of the facility, whereas a 20-year operating period is used in other Phase 4A site selection studies. In the case of short term releases, exposures were based on the most hazardous waste stream handled at the facility and an exposure period equal to the duration of the release.

The predicted chemical concentrations in air, cistern and pond water, beef, milk and leafy vegetables were then used as input to the final step of the exposure assessment: the calculation of the daily human dose obtained from various multimedia exposures. The dose calculations utilized standard dietary and metabolic assumptions for an average man doing light work as shown in Table 4. While more severe assumptions for the exposed populations could have been made, e.g., children, or adults engaged in heavy work, these were not adopted since they were considered less representative of the general population surrounding the candidate sites.

HAZARD EVALUATION

The hazard evaluation component of the OWMC risk assessment generated a series of toxicity profiles for the chemicals selected for modeling. These profiles are presented in Appendix A (see Volume 2 of this report). Each toxicity profile was prepared to reflect the type of exposure predicted for that chemical, i.e., chronic or acute (as described in Chapter 3). For chronic exposures to known carcinogens, the Unit Cancer

TABLE 4

STANDARD ASSUMPTIONS FOR HUMAN DOSE CALCULATIONS

Body weight, kg	70[a]
Breathing rate, m^3/d	23[b]
Water consumption, L/d	2.0[c]
Leafy vegetable consumption, kg/d	0.046[d]
Beef consumption, kg/d	0.139[e]
Milk consumption, kg/d	0.383[e]

Notes:

(a) National Institute for Occupational Safety and Health (NIOSH). 1980. Registry of Toxic Effects of Chemical Substances. 1979 Edition. Volume 1. DHHS (NIOSH) Publication No. 80-111.

(b) Altman, P.L., and Dittmer, D.S., eds. 1972. Biology Data Book, Second Edition. Federation of American Societies for Experimental Biology, Bethesda, MD. [Value used represents a man doing light work for ten hours (1.7 m^3/h) and at rest for 14 hours (0.4 m^3/h)].

(c) United States Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria Documents. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. (e.g., Ambient Water Quality Criteria for Phthalate Esters, EPA/440/5-80-067, PB81-117780).

(d) National Research Council of Canada (NRCC) 1979. Effects of Cadmium in the Canadian Environment. Publication No. NRCC 16743 Ottawa, Canada.

(e) United States Environmental Protection Agency (USEPA). 1978. Update of Food Factor Tables. Memo dated May 1, R.D. Schmitt, Toxicology Branch.

Risk (UCR) values used in the risk assessment were taken from the assessments prepared by the United States Environmental Protection Agency's Cancer Assessment Group (CAG, 1985). For chronic exposures to chemicals not known to produce carcinogenic effects, Acceptable Daily Intake (ADI) values were adopted from the values published by various program offices of the United States Environmental Protection Agency (USEPA, 1984b). In the absence of published values, ADI values were estimated from animal data using the methodology described earlier in this chapter (see page 16).

Acute exposures were evaluated in terms of the dose or concentration resulting in no effect as well as the dose or concentration resulting in no adverse effect on an exposed human population. For exposures by inhalation, consideration was given not only to the importance of chemical concentration, but also to the time dependence of toxicity.

RISK CHARACTERIZATION

Risk characterization of the releases from the OWMC facility was undertaken separately for (1) chronic exposure to carcinogens; (2) chronic exposure to non-carcinogens; and (3) acute exposures. In making the distinction betwen carcinogens and non-carcinogens, consideration was given to the particular route of exposure, since some chemicals, e.g., formaldehyde, may be carcinogenic by inhalation and not by ingestion. Chronic risk was characterized for carcinogens by multiplication of the calculated daily human dose (DHD) by the unit cancer risk (UCR). We note that the cancer risks presented here are for incidence of cancer which should not be considered equivalent to death.

$$\text{Cancer Risk} = (\text{UCR})(\text{DHD})$$

These numbers were then summed for all the carcinogens of concern and a total cancer risk for a particular pathway was derived. Total cancer risks for fugitive and incinerator emissions were calculated separately.

Non-carcinogenic risks were treated in a different manner. Non-carcinogenic risk for chronic exposures was obtained by dividing the Acceptable Daily Intake (ADI) for the chemical by the calculated daily human dose (DHD) in order to obtain an adjusted margin of safety (AMOS).

$$\text{Adjusted Margin of Safety (AMOS)} = \frac{\text{ADI}}{\text{DHD}}$$

The adjusted margin of safety developed for this study differs from the margin of safety described previously (see page 18) by incorporating a safety factor to derive the ADI from the No-observed-adverse-effect-level (NOAEL) for chronic exposure, i.e., ADI = NOAEL/Safety Factor. The AMOS values derived in this way were then grouped and aggregated for each organ of the body affected using the following formula:

$$\text{Aggregated AMOS} = (\Sigma 1/\text{AMOS})^{-1}$$

where Σ represents the sum of all $\frac{1}{\text{AMOS}}$ values for the same organ.

For example, all liver toxin risks were aggregated in this way. This allowed an estimation of the daily burden placed on an organ due to exposure from several chemicals.

These aggregation methodologies are consistent with guidelines for health risk assessment for chemical mixtures recently proposed by the United States Environmental Protection Agency (USEPA, 1985). In the absence of data to the contrary, the USEPA has suggested that risks from exposure to mixtures of chemicals be estimated as though their effects were additive. This approach is considered to provide a reasonable estimate of risk for the purposes of the generic risk assessment.

For acute exposures, the characterization of risk was conducted in a similar manner. In this case, however, an acute NOEL to which a safety factor had been applied was used (instead of an ADI) for comparison to the human dose (HD) received over a short time period.

$$\text{Adjusted Margin of Safety (AMOS)} = \frac{\text{(NOEL)}}{\text{(HD) (Safety Factor)}}$$

In some cases, depending upon the toxicological action of the chemical, it was more appropriate to make a comparison between a predicted concentration and a NOEL based on concentration rather than dose.

It should be emphasized that the use of a NOEL rather than a NOAEL in the characterization of acute risks represents a highly conservative approach. For short term exposures that occur relatively infrequently, a more conventional approach would be to base an acceptable level on the possible occurrence of minor, reversible and nonincapacitating effects (NRC, 1979). The more conservative approach to estimating AMOS values, however, was adopted as being consistent with the use of conservative assumptions throughout the risk assesment and OWMC's goal of minimizing risk to human health.

In assessing the risks resulting from the predicted exposures, it was necessary to evaluate the significance of the calculated AMOS values and cancer risks. For non-carcinogenic risks, exposure events that led to AMOS values of one or greater were considered not to pose any public health risk. In such cases, the ADI for chronic exposures, or the NOEL to which an appropriate safety factor had been applied for acute exposures, were greater than the predicted dose. Thus, the level of exposure was considered below that sufficient to cause an adverse health effect.

If the AMOS value was less than one, this result was interpreted differently for chronic and acute exposures. For chronic exposures, if the AMOS value was less than one, the predicted exposure was considered significant and so was the potential for human health effects.

For acute exposures, if the AMOS value was less than one, this was considered an indicator of a potential effect but not necessarily an adverse health effect, because of the use of the NOEL rather than NOAEL. In such cases, the potential for an adverse health effect was evaluated further using the criteria developed by the National Academy of Sciences (NRC, 1979) for short term exposure limits for air pollutants (see page 19). Using these criteria, the National Academy of Sciences (NAS) has currently put forward two types of short term exposure limits: short term public limits (STPLs), and emergency exposure limts (EELs)[2]. The short term public limits (STPLs) relate to predictable single exposures, usually lasting 60 minutes or less, but never more than 24 hours. If not exceeded, the STPLs are expected to be associated with, at most, nonincapacitating, reversible effects. They differ from the emergency exposure limits (EELs) in that the EELs were (1) developed initially for defined occupational groups, e.g., military personnel; (2) relate to unpredictable single exposures; and (3) reflect the likely occurrence of nonincapacitating, reversible effects while avoiding any significant decrement in motor performance during emergencies. The STPLs were considered more applicable to the objectives of the OWMC risk assessment and were used, therefore, to assess the potential for acute health effects in cases where the AMOS values were less than one.

---

[2] A third limit, termed the short term public emergency limit, was developed by NAS initially, but is not particularly useful in this context.

For carcinogens, the potential for public health risk was evaluated using a different format. For each risk pathway, a quantitative carcinogenic risk was calculated and the level of risk was evaluated against some level of acceptable risk. For the purposes of this risk assessment, a value of one in a million additional cancers over a lifetime of exposure was considered as the criterion for acceptable cancer risk. The use of this value finds precedent in the regulation of toxic chemical exposures in other situations.

For example, the United States Food and Drug Administration (USFDA), in its regulation of carcinogenic animal feed additives, established an acceptable residue level in the tissues of animals going to slaughter equal to or less than that level, which over a lifetime of exposure, would lead to an increased risk of one in a million additional cancers. Thus, the USFDA chose the one in a million risk level as a proper balance between the benefits derived from using carcinogenic animal feed additives and the potential public health risk that results from the consumption of residual levels of these contaminants in the food supply (USFDA, 1979).

Other risk values have also been used by governmental agencies in the United States in their efforts to regulate human exposure to carcinogenic agents. In establishing water quality criteria, the United States Environmental Protection Agency (USEPA) acknowledged the fact that, to insure the maximum protection of public health, no exposure to chemical carcinogens would be desirable. However, such an exposure limit is unobtainable and, therefore, the Agency derived quantitative estimates of cancer risk that could result from exposure to the chemicals of concern and expressed a range of risk estimates from 10^{-5} to 10^{-7} (USEPA, 1980). Similarly, in its proposed guidance for establishing alternate concentration limits (ACL)[3] for hazardous constituents in groundwater at hazardous waste sites, consideration is given to setting acceptable risk levels: "In general, the Agency has made decisions to allow concentrations of carcinogens where the individual risk values have been within the range 10^{-4} to 10^{-8}" (USEPA, 1984). The guidance recommends "that [RCRA] permit writers encourage owners and operators [of hazardous waste management facilities] to consider a level of 10^{-6}, the middle of the range, to be used as the point of departure when proposing a risk level within the 10^{-4} to 10^{-8} range for their particular facility."

---

[3] Hazardous waste regulations promulgated under the United States Resource Conservation and Recovery Act require that concentrations of hazardous constituents in groundwater measured at the edge of the waste management area do not exceed background levels. Alternate concentration limits (ACLs) can be established based on demonstration of no potential effects on human health and the environment.

Based on the above examples, the choice of a value of one in a million additional cancers over a lifetime of exposure was considered conservative and appropriate for the present analysis.

CHAPTER 3

CHARACTERIZATION OF RELEASES

3.1 CHARACTERISTICS OF FACILITY

The proposed OWMC facility will consist of the following major components:

- Waste handling and storage
- Incinerator plant
- Physical/chemical treatment plant
- Solidification plant
- Engineered landfill.

Also included here as part of the waste management system that can potentially result in environmental releases is the transportation of hazardous wastes to the facility, even though the transport vehicles are not planned to be operated by OWMC.

These components and the associated transportation system will be designed for an initial capacity of 150 000 metric tons per annum (t/a), which would include capacity for the treatment of 120 000 t/a of inorganic wastes, and the incineration of 30 000 t/a of organic wastes. Provisions have been made in the current design work for expansion of the total facility to an ultimate capacity of 300 000 t/a should this be necessary. For purposes of characterizing releases from the facility, it was conservatively assumed that the facility will operate at the upper capacity limit of 300 000 t/a.

Each of the components of the OWMC facility is described in detail in the Status Report - Facility Design prepared by the engineering consultants (Monenco, 1985a).

WASTE ARRIVAL AT FACILITY

It is expected that all wastes will arrive at the facility in trucks of various types and sizes ranging from pick-ups to large multi-axle vehicles. Wastes generally will be containerized in three ways: drums or other types of containers; dump trucks or luggers; and tankers (tank trucks or tank trailers).

WASTE HANDLING AND STORAGE

Prior to appropriate treatment and disposal at the facility, all wastes first will be sampled for analysis and then transferred to intermediate storage facilities. The precise nature of the handling steps involved will depend upon the physical and chemical characteristics of each waste and the container type in which the waste is received.

INCINERATOR PLANT

A rotary kiln incinerator will be used to destroy organic wastes at high temperatures. At the maximum planned capacity of the facility (300 000 t/a), two separate incinerator units are envisioned. Combustion gas from the incinerator(s) will be cleaned by a flue gas treatment system which will not generate any liquid effluent discharge.

PHYSICAL/CHEMICAL TREATMENT PLANT

A physical/chemical treatment plant will be used to treat inorganic wastes and remove contaminants using proven physical and chemical processes. The physical/chemical treatment plant will also be used to recover oils from selected aqueous organic wastes using physical/chemical processes; recovered oils will be sent to the incinerator plant for destruction.

SOLIDIFICATION PLANT

The solidification plant will use cement and other reagents and additives. These will be mixed with the residues of incineration and physical/chemical treatment to immobilize the contaminants in a solid mass. This solid mass will be disposed of in an engineered landfill.

ENGINEERED LANDFILL

The engineered landfill will be used to contain the solidified residues in an environmentally safe manner using appropriate design and operating procedures. No untreated waste will be deposited in the landfill. Leachate generated in the landfill will be collected and analyzed, and will be treated if necessary.

3.2 MAJOR SOURCES OF RELEASES

In characterizing environmental releases associated with the facility operations, four sets of conditions were considered: (1) normal operating conditions, (2) upset conditions, (3) on-site accidents, and (4) off-site transportation accidents. Based on a review of the facility design and operating procedures, the principal sources of environmental releases were identified as waste handling and storage, incineration, physical/chemical treatment, and transportation accidents. The sources of air emissions and the chemicals of concern associated with these emissions are summarized in Table 5. Each class of environmental releases is described briefly below.

NORMAL OPERATING CONDITIONS

The OWMC facility has been designed to minimize releases of toxic pollutants. Under normal operating conditions, however, routine emissions will be associated with waste handling and storage, physical/chemical treatment and incinerator operation.

Waste handling and storage and physical/chemical treatment, give rise to fugitive emissions, including volatile organic chemicals. These emissions are generated as residual emissions from pollution control devices and from miscellaneous uncontrolled sources. The following sources of fugitive emissions were identified for the OWMC facility and are described in greater detail in the engineering consultants' report entitled Site Selection Process. Phase 4A: Selection of Preferred Site(s) - Engineering (Monenco, 1985b):

- Waste unloading and transfer
- Drum opening
- Drum transfer
- Storage tanks
- Dissolved air flotation (DAF) unit
- Emulsion breaking
- Routine spillage.

TABLE 5

SOURCES OF ENVIRONMENTAL RELEASES

Facility Operation	Source of Release	Chemicals of Concern
A. NORMAL OPERATING CONDITIONS		
1. Waste Handling and Storage	Fugitive Emissions	Volatile organics
2. Physical/Chemical Treatment	Fugitive Emissions	Volatile organics
3. Incinerator	Stack Emissions	Organics, toxic by-products, metals and acid gases
B. UPSET CONDITIONS		
1. Incinerator	Scrubber Failure	Acid gases
C. ON-SITE ACCIDENTS		
1. Waste Handling and Storage	Mixing of incompatible wastes	Hydrogen sulfide
	Tank farm fire	Volatile organics
D. OFF-SITE ACCIDENTS		
1. Transportation	Accidental spill	Volatile organics

For the incinerator operations, air pollutants may be generated from a number of sources: organic chemicals present in the waste feed, acid gases generated in the combustion process, metals present in the waste feed, and products of incomplete combustion. These products of incomplete combustion (PICs) are formed as a result of partial oxidation of chemicals in the waste feed due to the combination of molecular fragments outside the combustion zone. At present there is limited information on the mechanism by which these PICs are formed, and how their formation can be related to waste feed or incinerator operating conditions (see ORF, 1985a). In the absence of such information, consideration was given to a particular set of possible PICs: the dioxins and furans. While there is considerable uncertainty about the formation of dioxins and furans in the incineration of hazardous wastes, their presence has been detected in low concentrations in the exhaust gases from some hazardous waste incinerators (see ORF, 1985a; Monenco, 1985a; Oberg, 1984; Radian, 1984). Because of the particular concern with the toxicity of these compounds, dioxin emissions were included in the risk assessment.

FACILITY UPSET CONDITIONS

At any industrial facility, the potential exists for equipment failure or operator error to result in upset conditions. At this preliminary stage in the design of the facility, a detailed hazard analysis of potential upset conditions has not been undertaken. It is recommended that such a hazard analysis be included in the next stage of the Facilities Development Process to facilitate site specific risk assessment. Based on the current design concepts, a number of potential upset conditions have been identified by the engineering consultants (see Monenco, 1985b). These include failure of the carbon adsorption system used to control air emissions from the waste handling and storage units; failure of the incinerator scrubber which removes acid gases; and pH excursions during the treatment of cyanide wastes or in the precipitation of metals using sulfide. Emissions associated with failure of the carbon adsorption system and pH excursions were considered of minor significance relative to the other emissions, and were not addressed further in the risk assessment.

ON-SITE ACCIDENTS

As is the case for facility upset conditions, there is the potential for on-site accidents which can generate releases of hazardous constituents. It is possible to envision a wide range of such accident scenarios with different severities and probabilities. Consistent with the philosophy of adopting conservative assumptions throughout the risk assessment, the analysis of on-site accidents was focused on occurrences that would result in relatively severe consequences.

As is the case for facility upset conditions, a detailed hazard analysis of the facility has not yet been undertaken. However, based on the current design, the most severe accidents identified by the engineering consultants (see M.M. Dillon, 1984; M.M. Dillon, 1985a; Monenco, 1985b) were: (1) the mixing of incompatible wastes with subsequent generation of hydrogen sulfide gas; and (2) a fire in the organic waste tank farm resulting in the boiling of a tank containing solvent wastes.

OFF-SITE TRANSPORTATION ACCIDENTS

Transportation accidents can occur on the access routes to the OWMC facility. For the purposes of the risk assessment, a traffic accident was envisioned whereby a tank truck containing volatile organic wastes spills its entire contents on the access route to the facility. Spills involving smaller releases may occur more frequently, either from traffic accidents or as a result of equipment failure. Again, consistent with the philosophy of adopting conservative assumptions throughout the risk assessment, a scenario involving total loss of contents was selected for analysis.

3.3 SELECTION OF SURROGATE CHEMICALS

For the environmental releases under normal operating conditions, there is a multiplicity of different chemicals which may be released depending upon the waste feeds in any given time period. Similarly, for the episodic releases that occur as a result of upsets and accidents, the precise nature of the chemical released will depend upon the particular waste stream being handled at the time and location of the incident. To model quantitatively the release of all such chemicals clearly would not be a feasible task. The approach adopted, therefore, was to select a manageable number of surrogate chemicals that represent the most hazardous chemicals to be handled at the facility. This approach is consistent with the conservative philosophy adopted throughout the risk assessment process.

The starting point for the selection of surrogate chemicals was the waste characterization data base which has been developed to estimate the types and quantities of special wastes generated in Ontario (Canviro, 1985). This waste inventory has been categorized using the University of California at Davis (UCD) classification scheme, whereby waste streams are grouped in UCD categories according to similarities in chemical constituents and physical state (see OWMC, 1982). Each UCD category can contain many different waste streams. These UCD categories are further organized into broader waste classes based on generic similarities.

To select surrogate chemicals for the various types of environmental releases, a series of ranking schemes was developed. For the long-term releases, i.e., releases under normal operating conditions, the objective of the ranking scheme was to identify the most hazardous chemical within a particular UCD category and, thus, to generate a list of surrogates representing all UCD categories. For the short-term releases, i.e., releases associated with upset conditions and accidents, the objective of the ranking scheme was to identify the most hazardous chemical that could be released in a particular incident. Each ranking scheme generated a hazard index as an indicator of the degree of hazard of a waste stream. The hazard indices were based on parameters specific to each source of release and are summarized in Table 6. Additional details of the ranking schemes used in the surrogate selection process are provided in Appendix B (see Volume 2 of this report).

TABLE 6

SELECTION OF SURROGATE CHEMICALS

Facility Operation	Source of Release	Selection Parameters
A. NORMAL OPERATING CONDITIONS		
1. Waste Handling and Storage	Fugitive Emissions	Chronic toxicity, chemical quantity, vapor pressure
2. Physical/Chemical Treatment	Fugitive Emissions	Chronic toxicity, chemical quantity, vapor pressure
3. Incinerator	Stack Emissions	Chronic toxicity, chemical quantity[1]
B. UPSET CONDITIONS		
1. Incinerator	Scrubber Failure	Not applicable[2]
C. ON-SITE ACCIDENTS		
1. Waste Handling and Storage	Mixing of incompatible wastes	Not applicable[3]
	Tank farm fire	Acute toxicity, chemical quantity, heat of vaporization
D. OFF-SITE ACCIDENTS		
1. Transportation	Accidental spill	Acute toxicity, chemical quantity, vapor pressure

Notes:

[1] Selection parameters for incinerator stack emissions under normal operating conditions apply only to volatile organics. Acid gases, metals and dioxins/furans selected from review of empirical data.

[2] Acid gases defined as contaminants of concern by upset condition.

[3] Generation of hydrogen sulfide selected as event of greatest concern in previous study (M.M. Dillon, 1984).

We note that in several cases a surrogate selection scheme was not applied since the surrogates were essentially "preselected". For example, in the case of incinerator emissions, the acid gases, metals and dioxins/furans were preselected by review of empirical data. Similarly, in the case of mixing of incompatible wastes, the use of hydrogen sulfide was determined by the event chosen in earlier work for OWMC on the selection of a technical buffer zone (M.M. Dillon, 1984), which was subsequently incorporated by the engineering consultants (Monenco, 1985b).

For the release types to which the surrogate selection schemes were applied, each hazard index was based on three factors: the inherent hazard of the chemical; the potential for the chemical to be released to the environment; and the quantity of chemical available for release. In the case of releases under normal operating conditions, risk to human health is based on long term exposure, so that the chronic toxicity was used to define the inherent hazard. For accidents and upset conditions, acute toxicity was considered a more appropriate measure of inherent hazard because of the short term nature of the exposures.

The definition of release potential was based on a parameter describing the tendency of the chemical to escape into the atmosphere. For transportation spills and fugitive emissions associated with waste handling and storage, the parameter selected was vapor pressure; for the tank farm fire, heat of vaporization provided a measure of the energy required to vaporize the chemical; and for incineration, the chemicals were assumed to behave essentially uniformly under high temperature conditions, so no differentiation of their release potential was included.

The quantity of chemical was based on the concentration data and estimates of waste quantities developed from the Canviro data base (see Canviro, 1985).

3.4 ESTIMATION OF RELEASE RATES

Release rates for the selected surrogate chemicals were estimated using a combination of empirical data from comparable facilities (ORF, 1985a) and by engineering calculations (Monenco, 1985b). The methodology and data base for each major source of release are outlined below. A more detailed description of the methodology and a summary of the release rates for each surrogate chemical are presented in Appendix C (see Volume 2 of this report).

FUGITIVE EMISSIONS

Fugitive emissions of volatile organic compounds were estimated using engineering calculations developed by the engineering consultants (Monenco, 1985b). Data inputs to these calculations were based on the current design of the facility (Monenco, 1985a) and the available information on waste characteristics, primarily from the Canviro data base (Canviro, 1985). Attention was focused on those waste classes which were considered most likely to contain significant concentrations of volatile organics, specifically:

Class 1	Organic sludges and still bottoms
Class 2	Solvent and organic solutions
Class 3	Oils and greases
Class 4	Oil and water mixtures
Class 5	Organic and oil residuals
Class 7	Miscellaneous chemicals and products
Class 8	Paint and organic residuals
Class 9	Aqueous solutions with organics
Class 12	Pesticide and herbicide wastes
Class 13	PCB wastes
Class 14	Cleanup residuals

In the case of oily wastes (Class 4) treated in the physical/chemical treatment plant, data on volatile organic compounds in these wastes were not available in the Canviro data base. For these wastes, the concentrations of volatile organics were based on a data base developed in the United States for waste oils (Franklin Associates, 1983).

In calculating the emission rates, it was conservatively assumed that all wastes go through each of the handling and storage and treatment operations identified as emission sources, with the exception of PCB wastes. PCB wastes in bulk will be pumped directly from arriving tank trucks to storage tanks, without intermediate handling in the reception basins, so that there will be no PCB emissions from the reception basins.

INCINERATOR STACK EMISSIONS

Incinerator stack emissions of organic chemicals were based on a 99.99% destruction and removal efficiency for the surrogate chemicals in the waste feeds. Concentrations of surrogate chemicals in the waste feeds were based on the available information on waste characteristics in the Canviro data base (Canviro, 1985). Other emissions, i.e., metals, acid gases and dioxins/furans were based on empirical data compiled by ORF (1985a).

INCINERATOR SCRUBBER FAILURE

In the event of a flue gas scrubber failure due to a power failure, the facility design and emergency procedures provide for shutdown of feed to the incinerator immediately and emergency power to resume the supply of water to the scrubber in less than two minutes (Monenco, 1985b). The emergency water supply from a head tank would meet the gas quenching requirements within the two minute period, and would also provide partial scrubbing of the gas. Gas flow would decrease to about 10% of normal in four minutes. After two minutes, the concentration of contaminants in the emitted gas would be as normal, and the gas flow would continue to fall to 10% of normal. For estimating the emission rate under these conditions, it was conservatively assumed that power failure could result in scrubber failure for both incinerator units.

MIXING OF INCOMPATIBLE WASTES

Emissions of hydrogen sulfide gas as a result of incompatible waste mixing were based on a scenario developed in earlier work for OWMC on the selection of a technical buffer zone (M.M. Dillon, 1984) which was adopted subsequently by the engineering consultants (Monenco, 1985b). This scenario assumes that "operational and engineering controls would prevent the gross contamination of sulfide wastes with strong acids, and that the worst probable occurrence would be the accidental addition of a barrel of strong acid to sulfide wastes or vice versa". The accident case analyzed assumed that one 55 gallon drum of 4% sulfuric acid was mixed with sulfide wastes in the physical/chemical treatment plant building. The gas would be emitted first into the building and then via its ventilation system to the atmosphere at roof level.

TANK FARM FIRE

Tank farm fire emissions were also based on a conservative scenario developed in earlier work for OWMC in the selection of a buffer zone (M.M. Dillon, 1984), which subsequently was adopted by the engineering consultants (Monenco, 1985b). In this scenario it is envisioned that a 2500 gallon tank full of the selected surrogate chemical is exposed to a fire surrounding the tank. The contents of the tank are boiled off through the tank's pressure relief valve, and vented above the level of flame effect. Assuming that the tanks are uninsulated, the rate of boil-off, or the emission rate, was estimated by the method of the American Petroleum Institute (API, 1976) as:

$$Q = \frac{(21\ 000)\ (F)\ (Aw)^{0.82}}{\Delta H}$$

where Q = emission rate, lb/h

F = insulation factor = 1.0 for a bare vessel

Aw = wetted surface area of tank, ft^2

ΔH = heat of vaporization, Btu/lb

For the purposes of the risk assessment, the scenario was modified (based on the results of the surrogate selection scheme which showed that methylene chloride was more hazardous than 1,1,1-trichloroethane) to use methylene chloride as the surrogate chemical instead of 1,1,1-trichloroethane used in the original scenario.

TRANSPORTATION SPILL

The transportation spill scenario assumes that a 30 tonne tank truck carrying a load of liquid waste is involved in a collision on the local access road, resulting in tank rupture and release of the entire contents of the truck. The released liquid spreads across the road surface to form a pool from which the selected surrogate chemical, methylene chloride, is released to the atmosphere. Clearly, different circumstances prevailing at the time of the spill (e.g., nature of the tank rupture, configuration of road surface, shoulder or roadside ditches) will affect the behavior of the liquid pool. As a conservative representation of pool size, the geometry of the pool was assumed to be circular with the center of the circle coincident with the centerline of the road, and the diameter constrained by the width of the paved surface of the road (7.0 m). The emission rate was then based on the equation of Stiver and Mackay (1982):

$$Q = \frac{(K_g)\ (A)\ (P)\ (M)}{(R)\ (T)}$$

where Q = emission rate, g/s

K_g = the gas mass transfer coefficient, m/s

A = area of spill, m^2

P = vapor pressure of the chemical or mixture, Pa

M = molecular weight of compound, g/mole

R = gas constant, Pa m^3/mole °K

T = temperature, °K

The derivation of the gas mass transfer coefficient and adaptation of the emission equation to address mixtures of compounds are presented in Appendix C (see Volume 2 of this report).

The emission rate is sensitive to environmental parameters, specifically wind speed, which affects the mass transfer coefficient (K_g) and ambient temperature. For example, the emission rate at 0°C will be approximately three times lower than at 30°C. For the purposes of the risk assessment, emission rates were estimated conservatively by using a temperature of 25°C, considered typical of average summertime temperatures in Southern Ontario. A wind speed of 1 m/s was used to correspond to the atmospheric stability class F subsequently used to model atmospheric transport (see Gartner Lee Associates Limited, 1982 for discussion of atmospheric stability classes).

CHAPTER 4

DIRECT EXPOSURES TO FACILITY EMISSIONS

4.1 PATHWAY DEFINITION

Direct exposures to facility emissions can be divided into two groupings: (1) long term exposures to emissions associated with normal facility operating conditions; and (2) short term exposures associated with facility upset conditions and accidents. For purposes of the risk assessment, it was assumed that human exposure occurs continuously over the maximum time period plausible, i.e., over a possible 30-year life of the facility for long term emissions and for the actual duration of episodic emissions.[4] Moreover, consistent with the conservative approach used throughout the risk assessment, it was assumed that an exposed individual is located permanently at the point of maximum concentration. The location of this point will be different for different emission types, depending largely upon the height of the emission source. In the case of incinerator stack emissions, this location will be the point of impingement located about two kilometres from the facility. For emissions which are vented from the physical-chemical treatment plant, under upset conditions, the point of impingement will be located approximately 300 m beyond the perimeter of the facility buffer zone. At distances either closer or further from the point of impingement, pollutant concentrations will be lower and human exposures will be correspondingly reduced.

---

[4] As indicated previously (see page 25), a 30 year operating period was selected as a more conservative representation of the operating life than the 20-year period used in other Phase 4A site selection studies.

For other emissions occurring at ground level or close to ground level, e.g., fugitive emissions, the maximum concentration for off-site receptors will occur at a point on the perimeter of the facility buffer zone. For these emissions, pollutant concentrations will be lower at distances beyond the perimeter of the facility buffer zone, and human exposures also will be correspondingly reduced. The methodology for assessing these human exposures and characterizing risk is described in the following sections.

4.2 LONG TERM EXPOSURES

To predict human exposures, the ambient concentration levels were modeled using a Gaussian dispersion plume model. Modeling of average annual concentrations was carried out using the United States Environmental Protection Agency's Industrial Source Complex model (see ORF, 1985b for discussion of this model). This Gaussian model calculates concentrations at an array of receptors for a complete range of meteorological parameters, and the results are weighted according to the frequency of occurrence of these parameters over the year. Meteorological conditions typical of both the Halton/Peel and Niagara regions were used initially as inputs to the model. Since the predicted concentrations were consistently higher for the Niagara region, these conditions were used for the exposure assessment. Incinerator stack emissions were modeled assuming a 60 m stack height. Fugitive emissions were considered in two groupings: those emitted at ground level or close to ground level, and those vented from the roof of the physical-chemical treatment building at a height of 25 m.

In applying the dispersion models, different time-averaging periods were used for emissions of carcinogenic and non-carcinogenic chemicals. For carcinogens, the total lifetime dose is the determining factor in defining risk, so that maximum annual concentrations were used to estimate human exposure. For non-carcinogens, it was conservatively assumed that the maximum average concentration over a two week period represents chronic exposure. Using these assumptions, the dispersion factors expressed as the estimated maximum annual and maximum two-weekly ambient concentrations for a unit emission rate are presented in Table 7.

TABLE 7

ESTIMATED DISPERSION FACTORS
FOR LONG TERM EMISSIONS

Emission Source	Maximum Annual Concentration $(ug/m^3)/(g/s)$	Maximum Two-Week Concentration $(ug/m^3)/(g/s)$
Incinerator Stack Emissions	0.036	0.3
Fugitive Emissions (Ground level)	23.5	120
Fugitive Emissions (25 m)	1.93	8

Source: Ontario Research Foundation

From the estimated ambient concentrations, a daily human dose (DHD) was calculated using the following equation:

$$DHD = \frac{(E_n)(D_f)(BR)(AF)}{BW}$$

where DHD = daily human dose, mg/kg/d

E_n = emission rate for chemical n, g/s

D_f = dispersion factor, $(mg/m^3)/(g/s)$ [see Table 7]

BR = breathing rate, m^3/d [see Table 4]

BW = body weight, kg [see Table 4]

AF = absorption factor, dimensionless

Human health risks were then estimated using the methodologies for carcinogens and non-carcinogens described in Chapter 2. For carcinogens, the measure of health risk for each chemical was the cancer risk, given by:

$$\text{Cancer risk} = (UCR)(DHD)$$

where UCR = unit cancer risk, $(mg/kg/d)^{-1}$

For the non-carcinogens, the measure of health risk was the adjusted margin of safety for each organ of concern, given by:

$$AMOS = \frac{ADI}{DHD}$$

where ADI = acceptable daily intake, mg/kg/d

For each of the non-carcinogens, the AMOS values were all greater than one, thus predicting that an adverse health effect would not be experienced at these exposure levels. The effects of non-carcinogens were therefore not considered further. For carcinogens, since there is no threshold below which a prediction of "no adverse health effect" can be made, cancer risks were aggregated for all carcinogens. The estimated cancer risks for fugitive and incinerator stack emissions were 5×10^{-7} and 1×10^{-6} respectively. In both cases, the risks are of a magnitude which should not be considered a significant human health hazard.

4.3 SHORT TERM EXPOSURES

To predict short term exposures, the ambient concentrations were modeled using a Gaussian dispersion plume model (see ORF, 1985b for discussion of this model). A short term model was used to establish concentrations for ground based inversions in the case of ground level emissions, and for fumigation conditions in the case of stack emissions. These were considered "worst-case" meteorological conditions. The height of the emission point was modeled for each emission source as follows: incinerator scrubber failure was modeled for a 60 m stack height; the tank farm fire was modeled as a ground level emission; and the mixing of incompatible wastes was modeled for a vent on the roof of the physical-chemical treatment building at a height of 25 m.

In applying the dispersion models, different time-averaging periods were used to represent acute exposures depending upon the duration of the emission. The maximum half-hour average concentrations were used to represent acute exposures for the tank farm fire and mixing of incompatible wastes. In the case of the incinerator scrubber failure, the emission can be considered as a release of short duration. As a conservative approach, the release was modeled as a continuous release of two minute duration. This two-minute period was considered to approximate the maximum exposure time for a receptor at the point of maximum concentration. Using these assumptions, the dispersion factors expressed as the estimated maximum half-hour and maximum two-minute ambient concentrations for a unit emission rate are presented in Table 8.

TABLE 8

DISPERSION FACTORS FOR SHORT TERM EMISSIONS FROM FACILITY UPSETS

Emission Source	Maximum Half-Hour Concentration $(ug/m^3)/(g/s)$	Maximum Two-Minute Concentration $(ug/m^3)/(g/s)$
Incinerator Scrubber Failure	24	38
Tank Farm Fire	3550	Not applicable
Mixing of Incompatible Wastes	160	Not applicable

Source: Ontario Research Foundation

For both the incinerator scrubber failure and incompatible waste mixing, health risk was evaluated by comparing the predicted concentrations to a concentration-based NOEL. For the tank farm fire human dose for the actual period of exposure was calculated using the following equation:

$$HD = \frac{(E_n)(D_f)(BR)(AF)(t)}{BW}$$

where HD = human dose, mg/kg

E_n = emission rate for chemical n, g/s

D_f = dispersion factor, $(mg/m^3)/(g/s)$ [see Table 8]

BR = breathing rate, m^3/h [see Table 4]

BW = body weight, kg [see Table 4]

AF = absorption factor, dimensionless

t = exposure time, h

Human health risks were then estimated as the adjusted margin of safety (AMOS) using the methodology described in Chapter 2:

$$\text{AMOS} = \frac{(\text{NOEL})}{(\text{HD})(\text{Safety Factor})}$$

The AMOS values are presented in Table 9.

TABLE 9

RISK ESTIMATES FOR ACUTE EXPOSURES

Source of Release	Chemical of Concern	Duration of Exposure	AMOS
Tank Farm Fire	Methylene chloride	2.6 h	0.01
Mixing of Incompatible Wastes	Hydrogen sulfide	42 min	10
Incinerator Scrubber Failure	Sulfur dioxide	2 min	0.06
	Nitrogen oxides		2
	Hydrogen chloride		0.5
	Hydrogen fluoride		10

Notes: 1) Incinerator stack scrubber failure based on simultaneous failure of scrubbers on both incinerator units.
2) AMOS values presented for point of maximum concentration: perimeter of facility buffer zone for tank farm fire; approximately 300 m beyond facility buffer zone for mixing of incompatible wastes, and approximately 2 km from facility for incinerator scrubber failure.

For the tank farm fire and incinerator stack scrubber failure, AMOS values less than one were predicted. However, it should be recognized that, at this time, the possible mitigation of these events by modifications to the facility design or operations has not been included. It should be recognized also, that the derivation of the AMOS values for short term exposure relies on the use of conservative safety factors, which were developed to protect against any effect rather than adverse health effects (see page 29).

The significance of the AMOS value in terms of the potential for adverse health effects can be assessed further by application of the methodology that would be used conventionally in establishing short term limits for public exposure. This methodology was described in Chapter 2 of this report (see page 30), and generates short term public limits (STPLs) designed for predictable single exposures, usually lasting 60 minutes or less, but never more than 24 hours. The STPLs are limits that, if not exceeded, are expected to be associated with, at most, a nonincapacitating, reversible effect in an exposed population. A nonincapacitating, reversible effect is defined as one that will not cause a period of disability; will not impair one's vision, visibility, judgement, or ability to breathe; and will not interfere with one's ability to escape from the site of exposure. These STPLs are considered appropriate for assessing the infrequent short term exposures associated with a tank farm fire or incinerator scrubber failure. A discussion of the STPLs to each of these scenarios follows.

In the event that the scrubber of both incinerator units fails simultaneously, AMOS values of 0.5 and 0.06 are predicted for hydrogen chloride and sulfur dioxide respectively. For hydrogen chloride, the predicted ambient concentration is 3.5 ppm for a period of approximately two minutes. The National Academy of Sciences (NRC, 1971) has reviewed exposure data on hydrogen chloride and established guidelines for short term exposure of the public. They conclude that the chief effect of inhalation exposure to hydrogen chloride will be irritation of the moist mucous membranes of the upper respiratory tract. Prevention of the disagreeable and irritating odor of hydrogen chloride thus was used as the basis for establishing short term public exposure limits. They assumed that the level at which hydrogen chloride could be detected is between 1-10 ppm and that values greater than 5-10 ppm are disagreeable and irritating. Using this approach NAS established a series of STPLs for exposure periods from ten minutes to one hour. For the ten minute period, an STPL of 4 ppm was established. This level is marginally higher than the 3.5 ppm predicted value expected to persist for two minutes. However, since the magnitude of the irritating effects for hydrogen chloride is likely to be a function of both concentration and exposure time, no adverse health effects would be expected for a two minute exposure period. The only effects likely to occur would be slight odors or minor, reversible irritation effects experienced by sensitive members of the population.

For sulfur dioxide the predicted ambient concentration is 1.7 ppm for a period of approximately two minutes. The effects of sulfur dioxide exposure are similar to those for hydrogen chloride, in that it acts as an irritant to the mucous membranes of the respiratory tract and the conjunctiva of the eye. The National Academy of Sciences (NRC, 1984) reviewed the data available on sulfur dioxide exposure and established emergency exposure limits (EELs) for sulfur dioxide (but not STPLs). The EELs were established for defined occupational groups such as military personnel or space personnel, rather than the general public. They differ from the STPLs in the acceptance of a nonincapacitating decrement in performance during emergencies. The EELs established by NAS are for exposure periods from 10 minutes to 24 hours. For the 10 minute period, which is most applicable to the two minute scrubber failure scenario, an EEL of 30 ppm was established. To allow for the potentially greater sensitivity of individual members of the exposed population, e.g., asthmatics, and in order to define a no effect level, a short term public limit was derived for this study from the EEL by applying a ten-fold safety factor. This was considered to provide an adequate margin of protection for asthmatics and other respiratory compromised individuals. The derived STPL of 3 ppm is higher than the 1.7 ppm predicted value expected to persist for two minutes. Since the magnitude of the irritating effects for sulfur dioxide is likely to be a function of both concentration and time, no adverse health effects would be expected for a two minute exposure period. For sensitive populations, however, it is possible that minor, reversible effects, e.g., temporary tightness of the chest, might be experienced.

In the case of the tank farm fire, the predicted dose at the point of maximum exposure is approximately 380 mg/kg and the AMOS value is 0.01. A short-term public limit (STPL) can be calculated for methylene chloride using a methodology parallel to that described for hydrogen chloride and sulfur dioxide. Applying the criterion that exposure must result in, at most, reversible effects in the exposed population, "no effect on psychomotor and cognitive measures" was reported at a concentration of 1000 ppm for two hours. This can be compared to a chronic 8 hour per day Threshold Limit Value (TLV) of 100 ppm and a Short Term Exposure Limit (STEL) of 500 ppm for occupational exposure (ACGIH, 1985). These data suggest that the STPL should be between 1000 ppm and 100 ppm. To allow for the potentially greater sensitivity of individual members of the public, an STPL was derived, therefore, by applying a five-fold safety factor to the 1000 ppm two-hour exposure. Using the assumptions for estimating human dose described in Table 3 (see page 23), an STPL of 17 mg/kg was estimated.

This is still significantly less than the predicted dose of 150 mg/kg. Therefore, while the biological impact on which the AMOS is based is extremely mild (i.e., changes in auditory threshold response), at the predicted exposure levels potentially significant health effects would be experienced. It is difficult to estimate how severe these effects would be, since human data for acute exposures to methylene chloride are generally for levels lower than this. For example, various studies cited in the literature for estimated doses in the range 22 to 67 mg/kg report effects such as lightheadedness, difficulty with speech, motor decrements and decreased eye and hand coordination (USEPA, 1983). However, the lowest reported dose at which a human fatality has occurred is 500 mg/kg. It can be concluded, therefore, that potentially severe effects would be experienced for exposures to methylene chloride at the predicted level, although these are likely to stop short of death or incapacitating illness.

It should be recognized in reviewing the predicted health effects associated with episodic events that their frequency of occurrence may be relatively low. Comprehensive data are not available at this time to provide a rigorous analysis of the frequency for the short term exposure scenarios. Review of data from other comparable facilities, however, suggests that the tank farm fire, in particular, is a highly improbable event. For example, at the Kommunekemi Facility in Nyborg, Denmark, as of March 1983, the facility operators indicate that there have been only eight incidents in almost ten years of operation (Chemcontrol, 1983). One of these incidents involved an explosion and fire in a tank storing sludge destined for the incinerator. The fire was extinguished by Kommunekemi's fire brigade and reportedly no persons were injured. The fire was believed to have been caused by an accumulation of static electricity igniting a combustible vapor mixture within the tank. To eliminate the potential for this type of incident, tanks which could contain organic materials have been grounded to prevent buildup of static electricity, and an inert gas blanketing system has been installed to prevent the formation of a combustible vapor mixture. These same provisions will be incorporated into the design of the organic storage tanks at the OWMC facility.

CHAPTER 5

INDIRECT EXPOSURES TO FACILITY EMISSIONS

5.1 PATHWAY DEFINITIONS

In addition to the direct human exposure to facility emissions, the risk assessment also considered indirect exposure to toxic chemicals through the ingestion of food and water. Relevant exposure pathways included: ingestion of locally grown vegetables, consumption of beef and milk from locally raised cattle, and the use of cisterns and ponds for drinking water.

Specifically, the following pathways were examined based on long-term fugitive and incinerator stack emissions of metals and organic chemicals as characterized in Chapter 4:

- Ingestion of leafy vegetables exposed to emissions from the air, and chemical uptake from soil depositions;
- Ingestion of cistern and pond water exposed to emissions from the air;
- Ingestion of beef and milk from cattle ingesting exposed vegetation and water.

The methodology used to characterize human health risks associated with these indirect pathways is outlined in the following sections. A more detailed description of the environmental fate and transport processes considered is presented in Appendix D (see Volume 2 of this report).

5.2 INGESTION OF LEAFY VEGETABLES

In order to model human exposure due to the ingestion of exposed locally grown leafy vegetables, several simplifying assumptions were made. First, it was not possible to model exposure from all crops potentially grown in the vicinity of the proposed facility. This limitation, however, was not considered a significant shortcoming. Since all plants do not accumulate chemicals to the same extent and leafy vegetables are considered to accumulate chemicals to a greater degree than non-leafy vegetables, a hypothetical leafy vegetable was conceptualized for the exposure analysis.

Second, it was assumed that an individual's average daily intake of leafy vegetable was solely from this exposed vegetation source, and that this source represented a 30-year (facility lifetime) supply. This conservative assumption would overestimate chemical doses likely to occur.

Third, a five-month growing season was assumed. The amount of chemical deposited on leaf surfaces and taken up from the soil by the plant is directly influenced by the length of time the plant is in the field. A five-month growing season, or period in the field, is a conservative estimate for leafy vegetables and also would overestimate chemical doses likely to occur.

Estimation of the dose received from the ingestion of exposed leafy vegetables necessitates the calculation of the amount of chemical accumulated by the plant. Plants can accumulate chemicals in three major ways: by uptake from the soil through the root system, by uptake from the air through stomata, and by direct surface deposition. Of these three modes of accumulation, no information is available on plant uptake from the air for the organic chemicals of concern and this pathway was therefore omitted by necessity in the analysis. The two remaining routes differ in importance depending on the nature of the chemical. For example, deposition onto a leaf surface is a more important phenomenon for metals and non-volatile organic chemicals such as PCBs than it is for volatile organic chemicals, due to the tendency of volatile chemicals to partition into the air. The concentration or accumulation of a chemical by the plant can thus be broadly defined by three variables: an uptake factor, a surface deposition factor, and the deposition rate of the chemical from the emission of concern (Holton et. al., 1984):

$$C_p = d[A_d + U_p]$$

where: C_p = chemical concentration in the plant, ug chemical/kg plant

d = chemical deposition rate, ug/m^2 s

A_d = leaf surface deposition factor, m^2 s/kg

U_p = soil uptake factor, m^2 s/kg

The significance of these variables and methods for their estimation are summarized below.

CHEMICAL DEPOSITION RATE

Chemicals in the atmosphere can come into contact with soil and plant surfaces through wet and dry deposition processes. Dry deposition describes the contact of very small particles and gases in the atmosphere, with a surface, due to atmospheric turbulence and molecular motion. The wet deposition process describes the contact of chemicals in the atmosphere with a surface due to washout by precipitation. The deposition rate used to determine the concentration of pollutant in the plant was obtained from the sum of dry and wet deposition processes.

LEAF SURFACE DEPOSITION

Leaf surface deposition plays a significant role in plant accumulation depending on the fraction intercepted by vegetation, crop yield and various chemical specific surface degradation processes. The extent of surface coverage of the plant is determined by the interception fraction of the plant and the crop yield. Baes et al. (1984) have calculated a theoretical maximum interception fraction for leafy vegetables for a unit area based upon planting practices, relative importance of the crop (by area planted) and time in the field. This calculated maximum value was used in estimating leaf surface deposition. To allow for the effect of crop yield, which has the effect of "diluting" the pollutant concentration in the plant by lowering the surface deposition factor, data on crop yields in Ontario were obtained from Ecologistics for asparagus, lettuce, cabbage, broccoli and cauliflower and combined for modeling of the hypothetical leafy vegetable.

For chemicals deposited on the surface of a leaf, two mechanisms for degradation were considered: volatilization and washoff by precipitation. For volatilization, only organic chemical volatilization was modeled; metals deposited onto leaf surfaces in the form of oxides or chlorides would not be lost through volatilization due to their extremely low vapor pressures. In the case of dioxin, no volatilization was assumed, since dioxin deposited onto a leaf surface may be strongly adsorbed to fly ash. This is a conservative assumption for a semivolatile chemical such as dioxin.

For precipitation, a weathering half-life of 15 days was assumed (Willis et al., 1980) for all the chemicals modeled from which a weathering rate constant was derived. This half-life was based on leaf washoff of the pesticide endrin, a highly hydrophobic chemical which would be expected to adsorb strongly to soil or leaf surfaces.

UPTAKE FROM THE SOIL

Chemicals deposited on the soil surface can leach into the soil solution in the root zone and be taken up by the plant. The extent of plant uptake was estimated using a plant-soil partition coefficient, with consideration also given to competing soil degradation processes. Soil-plant partition coefficients are defined as the ratio of the concentration of chemical in the plant to the concentration of the chemical in the soil, at equilibrium.

The principal sources for estimating these coefficients were the microcosm studies of Figge et al. (1983) and food chain risk assessment work undertaken by the US Army Medical Bioengineering Research and Development Laboratory (Rosenblatt and Small, 1981). The highest partition coefficient cited in these studies was conservatively selected for all chemicals in the risk assessment.

To allow for soil degradation processes, consideration was given to volatilization, leaching and biodegradation. Volatilization upward from the root zone through soil pores was estimated by the method of Bomberger et al. (1982). It was considered that such losses could be significant in removing chemicals of high volatility which do not adsorb onto soil particles. In the case of dioxin bound to fly ash, no volatilization was assumed.

Leaching from the soil depends on the amount of the precipitation, the soil porosity and the tendency of the chemical to resist migration by adsorption onto organic carbon found in the soil. This tendency is expressed by a partition coefficient (Koc), and describes the distribution of a chemical between soil organic carbon and water. It is therefore evident that soils with a large fraction of organic carbon will have a greater tendency to retain certain organic chemicals than soils with a low fraction of organic carbon. In order to calculate a leaching loss rate constant, it was assumed that the soils in question contain 1% organic carbon, which represents a typical soil organic carbon concentration (Gartner Lee Associates).

Biodegradation in soils is highly variable. Rate constants were obtained from the literature and where a range of values was indicated, the most conservative number (lowest rate of biodegradation) was selected. In the case of dioxin bound to fly ash, no biodegradation was assumed.

5.3 INGESTION OF BEEF AND MILK

The analysis of potential human exposure via ingestion of beef and milk was restricted to cattle since ruminants are good indicators of impacts on other species such as chickens and game. In order to assess the daily dose of chemical obtained from consuming contaminated beef and milk, it was first necessary to estimate the concentration of the chemical found in beef and milk due to leaf surface deposition and plant uptake. The model developed by Holton et al. (1984) was used to estimate this concentration by the use of cattle dietary assumptions, the concentration of chemical found in cattle food crops, and the fraction of chemical retained in beef and milk resulting from the consumption of exposed food crops:

$$C_1 = (F_1)(Q_g)(C_g)$$

where C_1 = the estimated concentration of the contaminant in milk or beef (ug/kg)

F_1 = fraction of the contaminant consumed each day which is transported and retained in milk or beef (d/kg)

Q_g = the quantity of grains and grasses eaten by cattle each day (kg/d)

C_g = estimated concentration of contaminant in grains and grasses (ug/kg).

The fraction of chemical retained in cattle after consuming exposed grain, hay and corn silage (F_1) was estimated using correlation equations developed by Holton et al. (1984).

The concentration of the chemical in cattle food crops (C_g) was modeled in the same manner as for leafy vegetables (see Section 5.2). The diet of the cattle was considered to be comprised of corn silage, mixed grain and hay and the quantity consumed per day (Q) was provided by Ecologistics.

An additional assessment was performed to determine the contribution to human health risk from cattle drinking contaminated pond water as well as consuming contaminated grain, again using water consumption data provided by Ecologistics. The concentration of the chemical in the pond water was determined as discussed in Section 5.4.

5.4 INGESTION OF CISTERN AND POND WATER

The impact of fugitive and stack emissions on the quality of pond and cistern water was assessed as follows. For fugitive emissions, organic chemical contamination was considered; while both organic chemical and metal contamination were considered for stack emissions. For the purpose of this analysis, the effects of acid gases were considered to be negligible.

The chemical concentrations in water were conceptualized as being controlled by two different processes depending upon whether organic chemicals or metals are being considered.

The concentration of organic chemicals in pond and cistern water due to fugitive and stack air emissions was determined by Henry's Law (Lyman et al, 1982). Henry's Law describes how a gaseous chemical will distribute itself between air and water under ideal conditions:

$$C_A = (C_s)(H)$$

where C_A = equilibrium air concentration of the chemical above the dilute aqueous solution, g/cm^3

C_s = equilibrium aqueous concentration (dilute) of the chemical, g/cm^3

H = Henry's Law constant, dimensionless

Henry's Law cannot be used to estimate metal concentrations in pond and cistern water. This is because metals emitted from incinerators will not remain as a gas, but will quickly adsorb to fly ash or exist as solid oxides. For particles, therefore, concentrations must be estimated by the mass of material deposited into a volume of water.

Ponds in the vicinity of the candidate sites are typically 0.6 m to 7.6 m in depth (Gartner Lee Associates). The long term metal concentration in such a pond can be expressed as:

$$C_m = \frac{d(K_s)}{P-E}$$

where C_m = concentration of metal in the pond, g/m^3

d = deposition rate, g/m^2 a

P = average annual precipitation, m/a

E = average annual evapotranspiration, m/a

K_s = sediment-water partition coefficient, dimensionless

The particulate deposition approach was used in estimating concentrations for all metals emitted from the incinerator. This method is considered conservative since it does not take into account any dilution afforded by the initial contents of the pond. As a conservative assumption, it was also assumed that the sediment-water partition coefficient, i.e., the ratio of the mass of chemical in solution to the mass of chemical absorbed on the sediment, would be one. A Henry's Law approach was applied for all organic chemicals, with the exception of dioxins and furans. Since dioxins and furans may be strongly adsorbed to fly ash emitted from the incinerator, both the Henry's Law and particulate deposition approaches were considered. Using both methods, the particulate deposition approach was found to result in the highest water concentrations for dioxins and furans. Consistent with the conservative approach used throughout the generic risk assessment, the higher value was adopted for assessing exposure.

5.5 EXPOSURE ASSESSMENT AND RISK CHARACTERIZATION

From the estimated ambient concentrations, a daily human dose was calculated using the following equations:

Leafy vegetables

$$DHD = \frac{(C_p)(Q_l)(AF)}{(BW)(1000)}$$

Milk

$$DHD = \frac{(C_i)(Q_c)(AF)}{(BW)(1000)}$$

Beef

$$DHD = \frac{(C_i)(Q_b)(AF)}{(BW)(1000)}$$

Pond and cistern water

$$DHD = \frac{(C_w)(Q_w)(AF)}{(BW)}$$

where DHD = daily human dose, mg/kg/d

C_p = chemical concentration in plant, ug/kg

C_i = chemical concentration in beef or milk, ug/kg

C_w = chemical concentration in water, g/m^3

Q_l = leafy vegetable consumption, kg/d

Q_b = beef consumption, kg/d

Q_c = milk consumption, kg/d

Q_w = water consumption, L/d

BW = body weight, kg

AF = absorption factor, dimensionless

1000 = factor for unit conversion

Human health risks were then estimated using the methodologies for carcinogens and non-carcinogens described in Chapter 2. For carcinogens, the measure of health risk for each chemical was the cancer risk, given by:

$$\text{Cancer risk} = (\text{UCR})(\text{DHD})$$

where UCR = unit cancer risk, $(\text{mg/kg/d})^{-1}$

For the non-carcinogens, the measure of health risk was the adjusted margin of safety for each organ of concern, given by:

$$\text{AMOS} = \frac{\text{ADI}}{\text{DHD}}$$

where ADI = acceptable daily intake, mg/kg/d

For each of the non-carcinogens, the AMOS values were all greater than one, thus predicting that an adverse health effect would not be experienced at these exposure levels. The effects of non-carcinogens were, therefore, not considered further. For carginogens, since there is no threshold below which a prediction of "no adverse health effect" can be made, cancer risks were aggregated for all carcinogens. The estimated cancer risks for fugitive and incinerator stack emissions are shown below in Table 10.

In each case, the risk is of a magnitude not considered to pose a significant human health hazard. Furthermore, the same conclusion can be reached for these exposure pathways when considered in combination, since the combined risks would be less than the one in a million criterion of acceptable risk.

TABLE 10

ESTIMATES OF LIFETIME CANCER RISK FOR

CHRONIC EXPOSURE FROM INDIRECT PATHWAYS

Exposure Pathway	Fugitive Emissions	Stack Emissions
Ponds and Cisterns	2×10^{-9}	4×10^{-7}
Crops	2×10^{-8}	3×10^{-7}
Livestock - Beef	3×10^{-11}	4×10^{-7}
Livestock - Milk	2×10^{-11}	2×10^{-7}

CHAPTER 6

EXPOSURE VIA TRANSPORTATION SPILLS

6.1 PATHWAY DEFINITION AND SPILL FREQUENCY

Direct exposure to toxic pollutants via the air pathway can occur as a result of a spill on the local access route if the spill occurs in proximity to a neighboring residence. Such spills can occur in the transportation of three groups of commodities associated with the OWMC facility: (1) special wastes transported to the facility; (2) reagents and additives transported to the facility; and (3) inter-site transfers of treatment residues in the event that the components of the facility are located at more than one site. An analysis of the relative risks associated with the transportation of these three commodity groups is presented in Appendix E. The analysis indicates that the risks posed by spills of special wastes are significantly greater than those posed by either reagents and additives or inter-site transfers. The generic risk assessment, therefore, was limited to spills of special wastes on the local access route.

Based on reconnaissance studies of the potential local and regional access roads (defined as that part of the access route between the 400 Series Highway and the facility), the closest residences were estimated to be 20-50 m from the centerline of the road in urbanized areas, and 30-160 m from the centerline of the road in the urban fringe and rural areas (M.M. Dillon). Consistent with the conservative approach adopted throughout the risk assessment, human exposure was assessed at the closest estimated distance of 20 m.

The dose received by a human receptor will be directly proportional to the exposure time and concentration. For a spill incident, the exposure time will be determined by one of two factors: the time needed for evacuation of potentially affected individuals from the zone of influence of the spill or the time for cleanup of the spill by an emergency response team. Both these responses are subject to variations, depending on the location and time of the spill, and the rapidity of spill reporting, as well as the location of emergency services. Because of these uncertainties, likely exposure times may vary from several minutes up to several hours. For the risk assessment, the exposure time was assumed to be one hour. This was considered a conservative assumption, given the relatively close proximity of the potential spill sites to the OWMC facility, where response teams and equipment will be located.

As indicated in Chapter 3, transportation spills can occur as a result of both traffic accidents and equipment failures. Traffic accidents were considered to represent a more severe spill scenario for the risk assessment. Unlike the episodic releases associated with facility operations, the data base for transportation accidents is relatively well documented and can be reasonably applied to hazardous waste transportation if the ratio of spills per accident is known. In a study prepared for OWMC (Pilorusso, 1983), a spill rate of 0.196 spills per truck accident was predicted, and was used for estimating spill rates for risk assessment. Using data for accidents on the local and regional access roads, the truck accident-related spill frequency for special wastes on the local and regional access roads has been defined by the transportation consultants (M.M. Dillon, 1985b) as:

$$S_F = (A_F)(f_{TG})(f_{SA})$$

where S_F = truck accident-related spill frequency

A_F = general vehicle accident frequency

f_{TG} = ratio of accident frequency for trucks to general vehicle accident frequency

f_{SA} = number of spills per truck accident.

These estimates are summarized for the siting options under consideration in Table 11. The spill rate associated with truck accidents was estimated to be 0.017 to 0.331 spills per year for all special wastes. This estimate, however, applies to the probability of a spill of any special waste being transported to the facility. The most hazardous wastes transported to the facility, which contain the surrogate chemical methylene chloride in high concentrations, are UCD categories 211 and 213. These waste categories represent approximately 2450 t/a or 0.8 percent of the projected 300 000 t/a ultimately to be received at the facility. If this factor is applied to the estimated spill frequency, the predicted frequency becomes one spill every 377 to 7353 years, depending on the length of the local and regional access road travelled (as shown in Table 11).

TABLE 11

FREQUENCY OF TRUCK ACCIDENT-RELATED SPILLS OF SPECIAL WASTES ON LOCAL AND REGIONAL ACCESS ROADS

Siting Option	Two-Way OWMC Annual Accident Involvement Frequency	Truck Accident-Related Spill Rate for All Special Wastes	Truck Accident-Related Spill Rate for Methylene Chloride Wastes
LF-9C	2.344	0.230	1.84×10^{-3}
LF-3C	3.380	0.331	2.65×10^{-3}
LF-1E	0.528	0.052	4.16×10^{-4}
LF-1E/PI-27E	0.172	0.017	1.35×10^{-4}
LF-4N/PI-1N	1.411	0.138	1.10×10^{-3}
LF-4N/PI-13N	1.109	0.109	8.72×10^{-4}

Notes: 1) Spill frequency based on 300 000 t/a special wastes transported to OWMC facility.

2) Spill frequency for methylene chloride wastes based on 0.8% total special wastes.

6.2 EXPOSURE ASSESSMENT AND RISK CHARACTERIZATION

To predict human exposure associated with a transportation spill, dispersion of the surrogate chemical emitted from the spill was estimated using a Gaussian plume model for a ground level source (Turner, 1970).

$$X = \frac{Q}{(\pi)(\sigma_y)(\sigma_z)(u)}$$

Where X = concentration in the air, g/m^3

Q = emission rate, g/s

σ_y = dispersion coefficient in the lateral direction, m

σ_z = dispersion coefficient in the vertical direction, m

u = windspeed, m/s

This model is designed for application to a point source emission. To apply it to the area source constituted by a pool of liquid, a correction is required to approximate the area source as a point source at some distance upwind from the actual spill site. This correction, referred to as the calculation of a virtual point, was estimated by the method of Turner (1970).

In applying the Gaussian plume model to the spill scenario, it was assumed that the topography in the vicinity of the spill site is essentially flat, and that there are no major perturbations introduced by the proximity of large buildings or other topographic irregularities. The atmospheric conditions assumed for dispersion were stability class F and a wind speed of 1 m/s. Class F reflects the most stable condition and thus accounts for the least amount of chemical dispersion (see Gartner Lee Associates, 1982 for discussion of atmospheric stability classes). Consistent with the approach of using conservative assumptions throughout the risk assessment, F stability was considered appropriate for modeling atmospheric dispersion.

An important component of the dispersion estimate was the choice of dispersion coefficients. These coefficients depend on the distance from the emission source, and are specific to the atmospheric stability class. The use of such dispersion coefficients in a Gaussian model is generally undertaken at distances of 100 m or more, since at shorter distances the behavior of the plume may be significantly influenced by mechanical mixing, particularly for a heavy vapor such as methylene chloride. This limitation was recognized in predicting atmospheric concentrations at distances less than 100 m. The approach adopted here was the use of extrapolated values for the lateral dispersion coefficient and a value of unity for the vertical dispersion coefficient.

From the estimated ambient concentrations, a human dose was calculated as:

$$HD = \frac{(X)(BR)(AF)(t)(1000)}{BW}$$

where HD = dose received, mg/kg

X = air concentration, g/m^3

BR = breathing rate, m^3/hr [See Table 4]

AF = absorption factor, dimensionless

t = exposure time, h

BW = body weight, kg

1000 = factor for unit conversion

Human health risk was then estimated as the adjusted margin of safety (AMOS) using the methodology described in Chapter 2:

$$\text{AMOS} = \frac{\text{HD}}{(\text{NOEL})\ (\text{Safety Factor})}$$

The AMOS value was estimated to be 0.005. This value is approximately two to three orders of magnitude less than one, suggesting the potential for severe health effects, similar to those described for the tank farm fire in Chapter 4 (see page 59), in the event of such exposures. In considering this result, however, it should be recognized that this level of severity is experienced for only 0.8 percent of the special wastes transported, and that these wastes are expected to be involved in a spill only every 377 to 7353 years. Furthermore, in practice the frequency of this scenario will be lower, since not all spills will result in a total loss of truck contents and a large pool, nor will all spills occur at a point on the local and regional access road adjacent to a residence within 20 m of the road.

It is instructive to extend this analysis to consider spills of other waste types that would result in an AMOS value less than one.

Approximately eight percent of all special wastes would fall into this category, and would be expected to be involved in a spill every 34 to 654 years. Thus, it can be concluded that approximately 92% of special wastes transported to the facility do not present any potential for effects via the air pathway in the event of a spill.

CHAPTER 7

CONCLUSIONS

7.1 GENERAL

In the previous chapters, specific risk pathways have been described through which human exposure can occur to toxic pollutants emitted from the operation of the OWMC facility. The predicted exposures have been evaluated in terms of their ability to cause both acute or immediate health damage, and long-term or chronic health damage.

For both fugitive and stack emissions, resulting from the normal operations of the OWMC facility, acute exposures were judged sufficiently low as not to contribute to acute health risk. Therefore, health risk associated with these emissions was evaluated in terms of lifetime cancer risks. In the case of accidents or facility upsets, the reverse was true. These episodic events do not contribute significantly to lifetime burdens of chemical exposure and thus do not significantly affect long-term cancer risk. The exposure levels that result immediately following one of these events, however, may be sufficiently high to cause acute health effects. Thus, in each of these cases, an acute health effect analysis was conducted. The results of both the acute health effects and chronic health effects analyses are summarized below.

7.2 ACUTE EXPOSURES

Risk estimates for acute exposures are presented in Table 12 in terms of the predicted AMOS value. The AMOS, or ratio between the level of exposure expected to result in no effect and the level of exposure predicted as a result of an accident or facility upset, provides an indication of the significance of the predicted exposure.

TABLE 12

RISK ESTIMATES FOR ACUTE EXPOSURES

Source of Release	Duration of Exposure	AMOS
Tank Farm Fire	2.6 h	0.01
Mixing of Incompatible Wastes	42 min	10
Incinerator Stack Scrubber Failure	2 min	
- Sulfur dioxide		0.06
- Nitrogen oxides		2
- Hydrogen chloride		0.5
- Hydrogen fluoride		10
Transportation Spill	1 h	0.005

If the AMOS value is one or greater, the predicted levels of exposure are sufficiently low to avoid the potential for an acute health effect. In cases where the AMOS value is less than one, this is an indicator of the potential for some level of effect, although this may not necessarily be an adverse health effect.

There are three cases in which the AMOS value is significantly less than one: the transportation spill, the tank farm fire and incinerator stack scrubber failure. However, it should be recognized that, at this time, the possible mitigation of these events by modifying the design or operation of the facility has not yet been included. In the case of the incinerator stack scrubber failure, the predicted AMOS values for hydrogen chloride and sulfur dioxide are 0.5 and 0.06 respectively. Based on the application of criteria for establishing short term public limits, no adverse health effects are expected to result from the incinerator scrubber failure, although slight odor or minor, reversible irritation effects might be experienced.

For the tank farm and the transportation spill, the predicted AMOS values are approximately two orders of magnitude lower than one. In both cases, methylene chloride was the surrogate chemical modeled as being released. Also, in both cases, the predicted dose was significantly higher than the short term public limit developed for evaluating adverse health effects. The exact severity of harm that might occur as a result of these exposures is difficult to predict. However, the analysis suggests that the levels of exposure that might result are clearly significant, although not sufficient to cause death or incapacitating illness.

It must also be emphasized that the predicted levels of exposure are based on a number of conservative assumptions used in formulating the exposure scenarios, so that the risk levels are likely overstated. Furthermore, these events are expected to occur infrequently. In the case of the transportation spill, the level of severity indicated by the AMOS value would only be experienced by 0.8% of the special wastes transported to the facility, and these wastes are expected to be involved in a spill only every 377 to 7353 years. Further analysis indicates that for most wastes (approximately 92%) no effects would be expected even if a spill of the type modeled was to occur. Thus, for 8% of the wastes, some level of effect, ranging from minor effects not considered adverse to health to potentially severe health effects, might be expected based on the conservative modeling performed.

For the tank farm fire, no data are available at this time to provide rigorous analysis of the frequency of occurrence. Review of information from other comparable facilities, however, suggests that the tank farm fire scenario is also a low probability event.

7.3 CHRONIC EXPOSURE

Lifetime cancer risk levels associated with chronic exposure to fugitive air or stack emissions are presented in Table 13. In each case, the conservative assumptions regarding exposure underlying these estimates have been presented in previous chapters. The results indicate that in no case did the predicted levels of cancer risk exceed the criteria for acceptability of one in a million additional lifetime cancers. However, while none of the predicted exposure scenarios lead to unacceptable long-term cancer risks, they should not be treated as equivalent. Rather, the analysis suggests that direct exposure to both stack and fugitive emissions are more significant than indirect exposure via the consumption of contaminated food or water.

TABLE 13

ESTIMATES OF LIFETIME CANCER RISK FOR CHRONIC EXPOSURE

Exposure Pathway	Fugitive Emissions	Stack Emissions
Direct Inhalation	5×10^{-7}	1×10^{-6}
Ponds and Cisterns	2×10^{-9}	4×10^{-7}
Crops	2×10^{-8}	3×10^{-7}
Livestock - Beef	3×10^{-11}	4×10^{-7}
Livestock - Milk	2×10^{-11}	2×10^{-7}

Several orders of magnitude difference in risk exist between exposure to direct fugitive and incinerator stack emissions and exposure to indirect fugitive emissions. In the case of indirect exposures to stack emissions, the levels of predicted risk are closer to those associated with either direct exposure to fugitive or stack emissions. This is a direct consequence of including in the risk assessment the formation and emission of dioxins during the incineration process. However, while the potential presence of dioxin emissions increases the levels of risk associated with indirect exposure to stack emissions, the overall pattern of these results suggests that greater health risks for stack emissions are associated with direct exposures than with indirect exposures.

In addition to performing comparisons between selected risk pathways, it is useful to evaluate the predicted risk levels by comparing them to risk levels associated with other human activities. Presented in Table 14 are the lifetime risks associated with a number of different phenomena. In some cases, the reported risks are associated with voluntary human activities, such as smoking or eating charcoal broiled meat. In other cases, the risks are related to occurrences which essentially are involuntary experience, such as being struck by lightning or receiving normal background radiation. Compared to any of these risk levels, it is evident that the health risks predicted for the operation of the OWMC facility, based on a series of conservative assumptions, are less significant than risks normally and routinely experienced.

7.4 RANKING OF RISK FACTORS

The outputs of the risk assessment, provided in Tables 12 and 13, can be used directly in the factor ranking process. In order to evaluate the significance of each of the risk values derived in the risk assessment, three variables -- severity, frequency, and mitigation -- must be considered.

Severity refers to both the magnitude of the risk created by exposure and the nature of the illness produced. For example, a cancer risk of 1×10^{-6} is clearly more severe than a risk of 1×10^{-11}. Likewise, irritation of the nasal passages is clearly less severe than incapacitating illness or death. Therefore, each effect related to exposure can be ranked in terms of the degree of severity.

TABLE 14

RISKS OF DEATH FROM VOLUNTARY AND INVOLUNTARY EXPOSURES

Source of Exposure	Lifetime Risk[a]
ACCIDENTAL RISKS OF DEATH[b]	
All home accidents	7.7×10^{-3}
Lightning	3.5×10^{-5}
Fire arms (accidents)	7×10^{-4}
Motor vehicle accidents	1.68×10^{-2}
Electrocution	3.71×10^{-4}
CANCER RISKS FROM RADIATION EXPOSURES	
Average medical diagnostic x-ray	1.4×10^{-3}
Background radiation exposure (average U.S., sea level)	1.4×10^{-3}
Frequent airline passengers - 4 hours/week (cosmic rays)	7×10^{-4}
Excess cancer risk due to living in masonry building rather than wood	3.5×10^{-4}
CANCER RISKS FROM COMMON CARCINOGENS	
Person sharing room with smoker	7×10^{-4}
One pint of milk per day (aflatoxins)	1.4×10^{-4}
1/2 lb charcoal broiled steak/week (cancer only; heart attack etc., extra)	2.1×10^{-5}
Average smoker (cancer only)	8.4×10^{-2}
(all effects)	2.1×10^{-1}
All cancers	1.96×10^{-1}

Source: E.A.C. Crouch and Wilson. (1984).

Notes: a) All statistics based on U.S. data

b) Lifetime accidental risks based on annual values for a sequence of several years.

The second variable is the frequency of occurrence. Some emissions that produce adverse health effects are episodic in nature. They occur with a greater or lesser frequency depending on the nature of the event being considered (for example, incinerator scrubber failure may occur more frequently than a tank farm fire). Other emissions occur on a continual basis, such as incinerator stack or fugitive emissions. Therefore, in the case of emissions that are episodic, the frequency of occurrence is an important variable to consider in factor ranking.

The third variable of concern is mitigation potential. Some of the adverse health consequences predicted by the risk assessment are the result of exposures which can be mitigated while others are not. The direct inhalation pathway, for example, is one for which no readily available mitigation strategy is available to prevent exposure. Conversely, the ingestion of contaminated water from ponds and cisterns can be prevented and, thus, the risk associated with this exposure precluded. The potential for mitigation, therefore, is a variable to consider in factor ranking.

Each of the risk pathways presented in Tables 12 and 13 were evaluated considering the three variables described above. The results of that evaluation for both the chronic and acute exposure pathways are presented below.

CHRONIC EXPOSURES

In the case of chronic emissions that lead to increased cancer risks, the frequency of occurrence is not a variable which affects the ranking of these pathways since all result from chronic or continuous exposures. However, the severity of impact and the opportunity for mitigation are clearly different for each of the pathways. Since cancer is the health end-point of concern, the severity variable cannot be used to distinguish between the type of health injury produced, but rather to distinguish the frequency of expected incidence of disease. Using this criterion, it is evident that direct exposure produces a more severe effect than any of the indirect exposure pathways. Furthermore, in the case of each of the indirect exposure pathways, steps can be taken to mitigate against exposure, while for direct exposure such steps are not readily available. Therefore, based on both severity and potential for mitigation, the direct pathway for chronic exposure ranks as more significant than any of the indirect pathways.

A distinction can also be made between different indirect pathways, particularly with regard to fugitive emissions. As noted above, the risks associated with indirect exposure to stack emissions are essentially equal and reflect consideration of the potential emission of dioxins from the incineration process. However, in the case of fugitive emissions, there are significant differences among the severities predicted for the indirect pathways. Exposures to contaminants through the ingestion of contaminated water from ponds and cisterns, or the ingestion of contaminated crops, produces a more severe impact in terms of cancer risk levels than the ingestion of livestock meat or milk (see Table 13 above). For all indirect pathways, the potential for mitigation exists and, therefore, this variable cannot be used to rank the relative significance of the different indirect pathways.

In summary for the chronic exposures, the results of the analysis of direct and indirect pathways suggest that in the factor ranking process greater weight should be given to the potential for direct exposures versus indirect exposures. In addition, within the indirect exposure group, for populations potentially affected by fugitive emissions, greater weight should be given to potential exposures to contaminants through the ingestion of contaminated water and crops than through the ingestion of contaminants in livestock meat or milk.

ACUTE EXPOSURES

The analysis of acute health risk pathways (see Table 12) suggests that distinctions between these pathways also can be made. The situation which produces the least concern is the mixing of incompatible wastes, since the AMOS value calculated for this accident condition is 10, which is significantly greater than the threshold value of one. In the other three cases (the scrubber failure, the tank farm fire, and transportation spill) for which the AMOS values are less than one, the significance of severity of effects, frequency of occurrence, and mitigation potential are considered further below.

In the case of the incinerator scrubber failure, while the AMOS values are less than one, application of criteria for establishing short term public limits indicates that no adverse health effect would result, although slight odor or minor, reversible irritation effects might be experienced. Information on frequency of scrubber failure events is limited, although they are expected to occur less infrequently than the tank farm fire or transportation spill events. Mitigation potential is limited by the short duration of the upset condition (2 minutes) which, however, contributes to the minor nature of the effects experienced by an exposed population.

For the transportation spill, the AMOS value is significantly less than one, and consideration of short term public limits for assessing adverse health effects suggests that potentially severe health effects could be experienced at the point of maximum exposure. Transportation spills, however, are expected to occur relatively infrequently; as discussed in Chapter 6 (see page 76), the predicted spill frequency corresponding to the AMOS value is one spill every 377 to 7353 years. In addition, the potential for mitigation exists so that the severity of health impact predicted as a result of a transportation accident can be reduced from that predicted here. For example, if rapid emergency response to a spill event occurs, the period of exposure can be reduced, thus reducing the potential for adverse health impact. Therefore, while certain transportation spill events are capable of causing significant adverse health effects, the frequency at which they occur is very low and there is the potential to mitigate against their effects.

The tank farm fire analysis presented in this report is premised on the release of methylene chloride as was the transportation spill accident. In this case, too, the AMOS value is significantly less than one, and consideration of short term public limits similarly suggests that potentially severe health effects could be experienced at the point of maximum exposure. No data are available on the frequency of such an event, although the probability is believed to be extremely low. As with the spill event, the potential for mitigation exists such that emergency response to the fire situation or evacuation may reduce the period of exposure. The tank farm fire can thus be considered to pose a concern similar to that of the transportation spill since both have the potential for significant adverse health effects, their frequency of occurrence is expected to be low, and there is the potential to mitigate against their effects.

In summary for the acute exposures, the most important differences in the acute exposures considered are in the severity of potential effects. In the case of the transportation spill and tank farm fire, potentially adverse health effects are predicted; for the incinerator scrubber failure, only minor reversible effects are predicted; and for incompatible waste mixing, no effects would be expected. While there are other differences among the acute exposures, i.e., in their frequency of occurrence and mitigation potential, the severity ranking is considered the appropriate order of their relative significance.

A final point of comparison to be considered is the relative importance of the acute and chronic exposure pathways. There is, however, no scientific basis on which to make such comparisons. Their relative importance depends on subjective judgements as to the significance of acute health effects caused by an event of known probability versus a known likelihood of increased incidence of long term disease, in this case cancer resulting from continuous exposure. Judgements of this sort are considered beyond the scope of this report, although the information provided here will facilitate such policy decisions by OWMC.

In conclusion, factor ranking is an important process in site selection. The information provided in this risk assessment document allows comparison between different risk pathways and permits rankings to be made based on severity of effect, frequency of occurrence, and mitigation potential. Such information is directly useful in the factor ranking process and, ultimately, in site selection.

GLOSSARY

Acceptable Daily Intake (ADI)	Estimate of the largest amount of chemical to which a person can be exposed on a daily basis which is anticipated to result in no adverse effects (usually expressed in mg/kg/day).
Acute Exposure	Exposure of short duration, as opposed to chronic.
Adjusted Margin of Safety (AMOS)	Estimate of the largest amount of chemical to which a person can be exposed which is not anticipated to result in effects (usually expressed in mg/kg/day) divided by the actual amount of human exposure.
Carcinogen	A substance that increases the risk of cancer.
Chronic Exposure	Exposure of long duration, as opposed to acute.
Conservative Assumption	Assumptions which are conservative in the sense of overestimating possible human health risk.
Dose	Measurement of the amount received by the subject, whether human or animal, per unit of body weight.
Dose-Response Evaluation	A component of risk assessment that describes the quantitative relationship between the amount of exposure to a substance and the extent of toxic injury or disease.
Dose-Response Relationship	The quantitative relationship between the amount of exposure to a substance and the extent of toxic injury produced.

Emergency Exposure Limit (EEL)	Limits applicable to unpredictable short-term exposures which, if not exceeded, may result in nonincapacitating, reversible effects, but avoid significant decrements in performance during emergencies (see SPTL).
Epidemiological Study	Study of human populations to identify causes of disease. Such studies often compare the health status of a group of persons who have been exposed to a suspect agent with that of a comparable non-exposed group.
Exposure	To be accessible to the influence of a chemical or chemical action.
Exposure Assessment	A component of risk assessment that involves describing the nature and size of population exposed to a substance and the magnitude and duration of their exposure. The assessment could concern past exposures, current exposures, or anticipated exposures.
Extrapolation	The estimation of a value beyond the known range on the basis of certain variables within the known range, from which the estimated value is assumed to follow.
Fugitive Emissions	Emissions generated by residual losses from pollution control devices and miscellaneous uncontrolled sources. Excludes incinerator stack emissions.

Hazard Analysis	The systematic identification of potential accidents and upset conditions of industrial facilities, and the quantitative analysis of their probability (using techniques such as "fault tree" analysis) and consequence. This terminology is applied typically in the chemical and nuclear industries, and should not be confused with "hazard evaluation" (see below).
Hazard Evaluation	A component of risk assessment which involves gathering and evaluating data on the types of health injury or disease (e.g. cancer) that may be produced by a chemical and on the conditions of exposure under which injury or disease is produced.
High-to-Low-Dose Extrapolation	The process or prediction of low exposure risks from the measured high exposure-high risk data.
Human Health Risk	The likelihood (or probability) that a given exposure or series of exposures to a toxic substance will damage the health of individuals experiencing the exposures.
Ingestion	Type of exposure through the mouth.
Inhalation	Type of exposure through the lungs.
Interspecies Extrapolation Model	Model used to extrapolate from results observed in laboratory animals to humans.
Leachate	Aqueous liquid generated by infiltration of rainwater or snowmelt into landfilled wastes.

Lifetime Exposure	Total amount of exposure to a substance that a human would receive in a lifetime (usually assumed to be seventy years).
Local and Regional Access Road	That portion of the access route between the 400 series highway and the facility.
Margin-of-Safety (MOS)	Maximum amount of exposure producing no measurable effect in animals (or studied humans) divided by the actual amount of human exposure.
Mathematical Model	Numerical procedure used during risk assessment to perform extrapolations.
No-Observed-Adverse-Effect-Level (NOAEL)	Dose level at which no adverse effects are noted.
No-Observed-Effect-Level (NOEL)	Dose level at which no effects are noted.
Potency	Amount of material necessary to produce a given level of a deleterious effect.
Products of Incomplete Combustion (PICs)	Chemicals formed as a result of partial oxidation of chemicals in the waste feed due to the combination of molecular fragments outside the combustion zone.
pH Excursion	Deviation from normal pH conditions during a chemical treatment process.
Risk	Probability of injury, disease, or death under specific circumstances.

GLOSSARY - Continued

Risk Assessment	The scientific activity of evaluating the toxic properties of a chemical and the conditions of human exposure to it, both to ascertain the likelihood that exposed humans will be adversely affected and to characterize the nature of the effects they may experience.
Risk Characterization	Final component of risk assessment that involves integration of the data and analysis involved in hazard evaluation and human exposure evaluation to determine the likelihood that humans will experience any of the various forms of toxicity associated with a substance.
Risk Management	Decisions about whether an assessed risk is sufficiently high to present a public health concern and about the appropriate means for control of a risk judged to be significant.
Risk Pathway	Describes pathway by which chemical release into the environment is transported to a human receptor.
Route of Exposure	Method by which the chemical is introduced into the biological organism, e.g. inhalation, ingestion, skin absorption, etc.
Safe	Condition of exposure under which there is a "practical certainty" that no harm will result in exposed individuals.
Safety Factor	Adjustments to the experimentally observed NOEL to compensate for uncertainties in the data and for imprecision in extrapolation to the general human population.

Scientifically Plausible	An approach or concept having substantial scientific suppport but is not completely certain.
Short Term Exposure Limit (STEL)	A 15-minute time-weighted average exposure which should not be exceeded at any time during a workday even if the 8-hour time-weighted average is within the TLV.
Short Term Public Limit (STPL)	Limits applicable to predictable single exposures of short duration which, if not exceeded, are expected to be associated with, at most, a nonincapacitating, reversible effect (see EEL).
Special Wastes	Industrial wastes that cannot be adequately dealt with by the existing public facilities such as municipal sewage treatment plants, municipal incinerators and municipal landfills.
Surrogate Chemical	Chemicals chosen to represent the most hazardous waste constituents to be managed at the OWMC facility.
Systemic Effects	Effects observed at sites distant from the entry point of a chemical due to its absorption and distribution into the body.
Teratogen	A chemical or agent that causes physical defects in the developing embryo after maternal exposure to the chemical.
Threshold Dose	The dose that has to be exceeded to produce a toxic response.

Threshold Limit Value (TLV)	Time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek, to which nearly all workers may be exposed repeatedly, day after day without adverse effect.
Toxic	Harmful in regard to the effects of chemicals.
Unit Cancer Risk (UCR)	Estimate of the lifetime risk due to each unit of exposure in the low exposure region.

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH). 1981. *Documentation of the Threshold Limit Values.* Fourth Edition. ACGIH, Cincinnati, OH.

American Petroleum Institute. 1976. *Recommended Practice for the Design and Installation of Pressure-Relieving Systems in Refineries. Part 1-Design*. API Recommended Practice 520. Fourth Edition.

Baes, G.F., Sharp, R.D., Sjoreen, A.L. and Shaw, R.W. 1984. *A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides in Agriculture*. Oak Ridge National Laboratory, Oak Ridge, TN (ORNL-5786).

Bomberger, D.C., Gwinn, J.L., Mabey, W.R., Tuse, D., and Tsong Wen Chou. 1982. *Environmental Fate and Transport at the Terrestrial-Atmospheric Interface*. Symposium on Models for Predicting Fate of Chemicals in the Environment. American Chemical Society, Division of Pesticides Chemistry, 184th National Meeting, Kansas City, MO.

Calabrese, E.J. 1983. *Principles of Animal Extrapolation*. John Wiley and Sons, New York.

CANVIRO Consultants, Ltd. 1985. *Market Assessment Program: Survey of 1000 Industrial Establishments*. Prepared for Ontario Waste Management Corporation.

Carcinogenic Assessment Group (CAG). 1985. *Relative Carcinogenic Potencies Among 54 Chemicals Evaluated by the Carcinogenic Assessment Group as Suspect Human Carcinogens*. In Health Assessment Document for Chlorinated Benzenes. USEPA Office of Health and Environmental Assessment. EPA/600/8-84/105F.

Chemcontrol A/S. 1983. *Eight Incidents at Kommunekemi Facility in Nyborg, Denmark*. Prepared for Environmental Elements Corporation, Baltimore, MD.

Crouch, E.A.C. and Wilson, R. 1984. *Inter-Risk Comparisons*. In Assessment and Management of Chemical Risks. Eds. J.V. Rodricks and R.G. Tardiff. ACS Symposium Series #239.

Figge, K., Klahn, J. and Koch, J. 1984. *A Kinetic Distribution Model for Chemicals Based on Results from a Standard Environmental System*. Fifth Annual Meeting of the Society of Environmental Toxicology and Chemistry, Arlington, VA.

Food Safety Council. 1980. *Proposed System for Food Safety Assessment*. Final Report of the Scientific Committee of the Food Safety Council. Washington, D.C.

Franklin Associates, Ltd. 1983. *Revised Draft Report on Waste Oil Management and Composition*. USEPA Office of Solid Waste.

Gartner Lee Associates Limited. 1982. *Primary Environmental Information. Part 4: Climatology - Volume I*. Prepared for Ontario Waste Management Corporation.

Gartner Lee Associates Limited. 1985a. *Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Geologic, Hydrogeologic and Geotechnical Considerations*. Prepared for Ontario Waste Management Corporation.

Holton, G.A., Travis, C.C., Etnier, E.L., O'Donnell, F.R., Hetrick, D.M. and Dixon, E. 1984. *Multiple-Pathways Screening-Level Assessment of a Hazardous Waste Incineration Facility*. Oak Ridge National Laboratory, Oak Ridge, TN (ORNL/TM-8652).

IRLG (Interagency Regulatory Liaison Group). 1979. Work Group on Risk Assessment. *Scientific Basis for Identification of Potential Carcinogens and Estimation of Risks*. Fed. Reg. 44:3985.

Klaassen, C.D. and Doull, J. 1980. *Evaluation of Safety: Toxicologic Evaluation in Casarett and Doull's Toxicology: The Basic Science of Poisons*. Second Edition. Edited by Doull, J., Klaassen, C.D., and Amdur, M.O. MacMillan Publishing Co., Inc., New York, pp. 11-27.

Lyman, W.J., Reehl, W.F. and Rosenblatt, D.H. 1982. *Handbook of Chemical Property Estimation Methods*. McGraw-Hill Book Co., New York.

M.M. Dillon Limited. 1984. *Site Selection Process. Phase 3B: Selection of Candidate Sites. Facilities Characteristics and Technical Buffer Zone.* Prepared for Ontario Waste Management Corporation.

M.M. Dillon Limited. 1985a. *Site Selection Process. Phase 4A: Selection of a Preferred Sites: Site Selection*. Prepared for Ontario Waste Management Corporation.

M.M. Dillon Limited. 1985b. *Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Transportation.* Prepared for Ontario Waste Management Corporation.

Monenco Limited. 1985a. *Status Report - Facility Design*. Prepared for Ontario Waste Management Corporation.

Monenco Limited. 1985b. *Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Engineering.* Prepared for Ontario Waste Management Corporation.

National Research Council. 1971. *Guides for Short-term Exposures of the Public to Air Pollutants*. I. Guide for Hydrogen Chloride. National Academy Press, Washington, D.C.

National Research Council. 1979. *Criteria for Short-Term Exposures to Air Pollutants*. National Academy of Sciences. Committee on Toxicology, Washington, DC.

National Research Council. 1980. Risk Assessment/Safety Evaluation of Food Chemicals. National Academy Press, Washington, D.C. 36 pp. Tomatis. "The value of long term testing for implementation of primary prevention." In Hiatt, H.H., Watson, J.G., Winsten, J.A., eds. Origins of Human Cancer. Book C. Human Risk Assessment. Cold Spring Harbor Laboratory. 1977.

National Research Council. 1980a. Drinking Water and Health. Volume 3. National Academy Press. Washington, DC.

National Research Council. 1984. Emergency and Continuous Exposure Limits for Selected Airborne Contaminants, Volume 2. Committee on Toxicology. National Academy Press, Washington, D.C.

Oberg, T. 1984. Health Risk Evaluation of Flue Gas Emissions of Chlorinated Dioxins and Dibenzofurans from a Hazardous Waste Incineration Plant. MKS Miljokonsulterna - Technical Note (translated from original in Swedish).

Ontario Research Foundation. 1985a. Emissions Related Studies, Preliminary Estimates of Air Emissions. Prepared for Ontario Waste Management Corporation.

Ontario Research Foundation. 1985b. Site Selection Process. Phase 4A: Selection of Preferred Site(s): Atmospheric Conditions. Prepared for Ontario Waste Management Corporation.

Ontario Waste Management Corporation. 1982. Facilities Development Process. Phase 1 Report. September.

Ontario Waste Management Corporation. 1985. Facilities Development Process. Phase 4A Report.

Pilorusso Research Associates Inc. 1983. Transportation of Special Wastes in Ontario. Prepared for Ontario Waste Management Corporation.

Radian Corporation. 1984. National Dioxin Study Tier 4 - Combustion Sources. Initial Literature Review and Testing Options. Prepared for United States Environmental Protection Agency, Office of Air Quality Planning and Standards. EPA-450/4-84-014b.

Rodricks, J.V. and Taylor, M. 1983. Application of Risk Assessment to Food Safety Decision-Making. Regulatory Toxicology and Pharmacology 3:275-307.

Rodricks, J.V. 1984. Risk Assessment at Hazardous Waste Disposal Sites. Hazardous Waste. 1(3)333-362.

Rosenblatt, D.H. and Small, M.J. 1981. Preliminary Pollutant Limit Values for Alabama Army Munition Plant. U.S. Army Medical Bioengineering Research and Development Laboratory.

Stiver, W. and Mackay, D. 1982. Spill Technology Newsletter, January/ February 1982. As cited in Technical Information for Problem Spills. Draft Introduction Manual.

Turner, D.B. 1970. Workbook of Atmospheric Dispersion Estimates. United States Environmental Protection Agency, Office of Air Programs.

Willis, G.H., Spencer, W.S. and McDowell, L.L. 1980. The Interception of Applied Pesticides by Foliage and their Persistence and Washoff Potential. A Field Scale Model for Chemicals Runoff and Erosion from Agricultural Management Systems. USDA Conservation Research Report No. 26.

United States Environmental Protection Agency. 1980. Criteria for the Protection of Human Health. Fed. Reg. 45:79323.

United States Environmental Protection Agency. 1981. Advisory Opinion for Dichloromethane (Methylene Chloride). Office of Drinking Water, Draft.

United States Environmental Protection Agency. 1983. Health Assessment Document for Dichloromethane (Methylene Chloride). Office of Health and Environmental Assessment, Washington, D.C. External Review Draft, EPA-600/8-82-004B.

United States Environmental Protection Agency. 1984. International Paper ACL Demonstration. Internal memorandum from Lee M. Thomas, Assistant Administrator to Morris Kay, Regional Administrator, Region VII.

United States Environmental Protection Agency. 1984b. Summary of Current Acceptable Daily Intakes (ADIs) for Oral Exposure. Internal Review Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. February 1984.

United States Environmental Protection Agency. 1985. Proposed Guidelines for the Health Risk Assessment of Chemical Mixtures. Fed. Reg. 50:1170-1176.

United States Food and Drug Administration. 1979. Chemical Compounds in Food Producing Animals: Criteria and Procedures for Evaluating Assays for Carcinogenic Residues. Fed. Reg. 44:17070-1714.

VOLUME 2

Technical Appendices

CONTENTS

Page

INTRODUCTION iii

APPENDIX A - TOXICITY PROFILES FOR SURROGATE CHEMICALS A-1

APPENDIX B - SELECTION OF SURROGATE CHEMICALS B-1

APPENDIX C - ESTIMATION OF ENVIRONMENTAL RELEASES C-1

APPENDIX D - DERIVATION OF ENVIRONMENTAL TRANSPORT DATA D-1

APPENDIX E - RELATIVE RISKS FOR TRANSPORTATION OF SPECIAL WASTES, REAGENTS AND ADDITIVES, AND INTER-SITE TRANSFERS E-1

INTRODUCTION

The following appendices provide supporting technical information to the Generic Risk Assessment documented in Volume I. The content of each appendix is summarized briefly below.

Appendix A - Toxicity Profiles for Surrogate Chemicals describes the methodology used to develop measures of toxicity for the surrogate chemicals used in the risk assessment. The purpose of the toxicity profiles was to determine acceptable or safe levels of exposure associated with the OWMC facility emissions. Profiles are presented for each of the surrogate chemicals documenting the known literature on toxic effects and concluding with the following data outputs:

- Unit Cancer Risks (UCRs) or Acceptable Daily Intakes (ADIs) for chronic exposures
- Acute Safe Exposure Levels (ASELs) and Short Term Public Limits (STPLs) for acute exposures.

Appendix B - Selection of Surrogate Chemicals describes the procedure used to select surrogate chemicals and estimate their concentrations in the organic waste streams to be managed by the facility. The selection process was designed to represent each category of wastes to be managed at the OWMC facility by the most hazardous constituent of the waste streams within that category. The appendix describes the ranking schemes used to select surrogates for the following sources of releases:

- Fugitive emissions
- Incinerator emissions
- Truck spill
- Tank farm fire.

The selected surrogates and the concentrations are presented, and the limitations of the selection procedure are described.

Appendix C - Estimation of Environmental Releases provides the basis for the estimation of emissions of gaseous pollutants associated with the facility under four possible scenarios:

- Normal facility operating conditions
- Facility upset conditions
- On-site accidents
- Off-site transportation accidents

The estimation methodology is presented for each of these four scenarios with the emphasis primarily on volatile organic chemicals. Emission estimates are included for each of the surrogate chemicals selected in Appendix B at facility capacities of 150 000 t/a and 300 000 t/a.

Appendix D - Derivation of Environmental Transport Parameters provides the supporting details for the models used to predict indirect exposures via ingestion of food and water. Atmospheric deposition rates are provided for each of the surrogate chemicals by both wet and dry deposition processes, and sub-models are described for the following phenomena:

- Leaf surface deposition
- Uptake from soil
- Retention in cattle tissues and milk

The output from the appendix is a summary of surrogate chemical concentrations predicted in leafy vegetables, cattle tissues and milk.

Appendix E - Relative Risks for Transportation of Special Wastes, Reagents and Additives, and Inter-site Transfers provides an assessment of the relative risks posed by a spill on the local or regional access route to the facility for the following commodities:

- Special wastes transported to the facility
- Reagents and additives transported to the facility
- Treatment residues transferred between units in the event that the components of the facility are located at more than one site.

The risks are compared in terms of both the consequence or severity of the commodity being spilled and the potential frequency of the event occurring; and a set of relative hazard indices are presented.

APPENDIX A

TOXICITY PROFILES FOR SURROGATE CHEMICALS

CONTENTS

Page

A-1 METHODOLOGY
- Chronic Acceptable Daily Intake A-1
- Unit Cancer Risk A-5
- Acute Safe Exposure Limit A-10
- Short Term Public Exposure Limit A-12
- Absorption Data A-15

A-2 TOXICITY PROFILES FOR ORGANIC COMPOUNDS
- Acrylonitrile A-19
- Benzene A-21
- Chloroform A-24
- Cresol A-26
- Dioxins/Furans A-28
- Ethanol A-33
- Formaldehyde A-35
- Hydrazine A-38
- Methyl ethyl ketone A-40
- Methylene chloride A-42
- Petroleum distillates A-52
- Phenol A-56
- PCBs A-57
- Pyridine A-59
- Styrene A-60
- Tetrachloroethylene A-62
- Tetraethyl lead A-66
- Toluene A-67
- 1,1,1-Trichloroethane A-68
- Trichloroethylene A-71

A-3 TOXICITY PROFILES FOR METALS
- Arsenic A-75
- Beryllium A-78
- Cadmium A-80
- Chromium (VI) A-83
- Copper A-85
- Lead A-87
- Mercury A-90
- Nickel A-92
- Zinc A-95

A-4 TOXICITY PROFILES FOR ACID GASES
- Hydrogen Chloride A-97
- Hydrogen Cyanide A-99
- Hydrogen Fluoride A-101
- Hydrogen Sulfide A-103
- Nitrogen Dioxide A-105
- Sulfur Dioxide A-107

REFERENCES A-111

LIST OF TABLES

		Page
A-1	Standard Values for Dose Conversions	A-6
A-2	Acceptable Daily Intake	A-7
A-3	Unit Cancer Risk	A-9
A-4	Short Term Limits	A-11
A-5	Summary of Inhalation Absorption Factors	A-16
A-6	Summary of Oral Absorption Data	A-17
A-7	Human Effects Reported After Short-term Exposure to Various Levels of Methylene Chloride	A-51

A-1 METHODOLOGY

This appendix provides a toxicity profile for each of the surrogate chemicals used in this report. Each profile provides an overview of the most pertinent toxicity data from which acceptable exposure limits were derived. Depending on the toxicological behavior of the compound and the exposure scenarios considered in the risk assessment, four different exposure limits were developed, as follows:

- Chronic acceptable daily intake (ADI)
- Unit cancer risk (UCR)
- Acute safe exposure limit (ASEL)
- Short term public exposure limit (STPL)

The methodology used to derive each of these limits is described in the following sections, which preceed the toxicity profiles. Information is also presented on the absorption of each chemical via the inhalation and oral routes of exposure. Each toxicity profile presents a review of the most relevant human and animal toxicity data, and then cites published ADI, UCR, ASEL, or STPL values. In cases where no published values are available, ENVIRON determined which of the toxicity studies provided the best and most relevant data, and then calculated an ADI, UCR, ASEL, or STPL.

CHRONIC ACCEPTABLE DAILY INTAKE (ADI)

For those chemicals not known to produce carcinogenic effects, the acceptable daily intake (ADI) is used as the measure of chronic toxicity. The use of the ADI as a measure of chronic toxicity has much precedent in toxicological science; ADIs are commonly used as the basis for establishing acceptable levels of human response to pesticide residues, drinking water contaminants, and food additives.

An ADI is defined by EPA as the amount of a toxic agent in mg/kg body weight/day (or in mg/d for a 70 kg person) which is not expected to result in any adverse effects after chronic exposure in the general population of humans, including sensitive subgroups (USEPA, 1980a). Operationally, ADIs are calculated by dividing the "no observed effect" level derived from animal or human toxicity studies by one or more uncertainty factors also called safety factors. In many cases, the only data available reflect "effect" levels, and various means have been developed to use such data for derivation of ADIs (USEPA, 1980a; NAS 1980).

Although exposure at the ADI is not expected to produce adverse health effects, the ADI is nevertheless directly related to the chronic toxic potential of a substance. Moreover, it is a measure that contains several systematic adjustments designed to yield a common measure of toxicity from diverse data sets.

ADIs have been established for a relatively large number of substances by various program offices of EPA, and these ADIs were adopted for the present ranking exercise without modification. The ADIs developed by EPA's

Environmental Criteria and Assessment Office (ECAO) (USEPA 1982 and 1984) and EPA's Office of Pesticide Programs (OPP) were used when available. ADI values reported in the National Academy of Sciences (NAS) series Drinking Water and Health were also used when EPA values were not available (NAS 1977). ADIs reported in units of mg/d (for a 70 kg person) were divided by 70 kg to yield ADIs in units of mg/kg body weight per day.

For substances for which ADI values have not been established by EPA or NAS, ADIs were estimated based on available human or animal toxicity data. The methods used for estimating ADIs are generally consistent with published EPA methods (USEPA 1980a). For chemicals for which toxicity data were extremely limited, however, it was necessary to introduce additional assumptions (which were incorporated as uncertainty factors) to derive the ADI. Details of the methodology used to estimate ADIs are presented below.

Methodology for Estimating ADIs

ADIs are generally calculated by dividing the no-observed-adverse-effect level (NOAEL) derived from animal or human toxicity studies by one or more uncertainty factors using the following formula:

$$\text{ADI} = \frac{\text{NOAEL}}{\text{Uncertainty Factor}}$$

The usual method of determining a NOAEL is first to identify the toxic response occurring at the lowest dose level in the most sensitive animal species for which test data are available. The highest dose level at which this toxic response is found to be unobservable is then identified; this is the no-observed-adverse-effect level or NOAEL. Preferably, the NOAEL used to derive the ADI is based on chronic toxicity data; if no chronic toxicity data are available, subchronic data are used with the application of additional uncertainty factors.

Recommended safety factors of 10, 100, 1000 were used to estimate ADIs depending on the available human or animal data (USEPA, 1980; NAS, 1977):

> A safety factor of 10 is used when the NOAEL is based on chronic human data. This factor is meant to account for intraspecies variability.
>
> A safety factor of 100 is used when the NOAEL is based on chronic animal data. This factor is meant to account for both intra- and interspecies variability.
>
> An safety factor of 1000 is used when the NOAEL is based on subchronic animal data. This factor incorporates uncertainty in extrapolating from subchronic to chronic exposures and includes the two previous uncertainty factors.

In cases where a NOAEL could not be identified, the lowest-observed-adverse-effect level (LOAEL) was used to estimate the ADI. In such cases, additional safety factors from 1-10 were used, depending on the severity of the effect observed. The intermediate safety factors were developed on a logarithmic scale (e.g., 3.2 being halfway between 1 and 10). The safety factors generally chosen were two and five representing approximately one-third and two-thirds, respectively, of the distance between one and ten. The safety factor of two was used in the case of relatively mild effects such as biochemical changes and potentially reversible, mild organ changes. The safety factor of five was reserved for cases where the effects were more severe such as organ necrosis, neurological effects, reproductive dysfunction and teratogenic effects.

In some cases, calculation of ADIs required dose conversion (e.g., from ppm in diet or mg/m^3 in air to estimate values in mg/kg body weight per day). The standard values for body weight, food consumption, water consumption, and air breathed used for dose conversion are listed below in Table A-1.

A list of all the ADI values included in this appendix is presented in Table A-2. Each potentially affected organ of the body is identified in the tabulation.

UNIT CARCINOGENIC RISK FOR CARCINOGENS

For those chemicals identifed as carcinogens, the unit carcinogenic risk (UCR) is used as the measure of carcinogenicity. The unit risk is defined as the upper limit on the lifetime probability that the carcinogen will cause cancer at a dose of 1 (mg/kg body weight)/day. The use of the term "upper limit" means that the actual risk is not likely to exceed the predicted risk, and that the actual risk is probably lower than the predicted risk and may even be zero (USEPA 1980). The unit risk is the slope of the carcinogenicity dose-response curve at low exposure levels, and, because of the way it is derived, it is constant over a wide range of low dose exposures. It is thus one systematic measure of carcinogenic potency and is suitable for ranking the relative potencies of various carcinogens in the low exposure region.

TABLE A-1

STANDARD VALUES FOR DOSE CONVERSIONS[a]

Species	Body Weight[b] (kg)	Food Consumption[c] (g/d)	Water Consumption[d] (mL/d)	Air Breathed[e] (L/min)
Mouse	0.025	3	5	0.03
Hamster	0.125	15	15	0.08
Rat:				
Adult	0.25	15	25	0.1
Weanling	0.05	15	25	0.05
Guinea pig	0.57	30	85	0.2
Rabbit	2	60	330	1
Cat[f]	2	100	--	0.32
Dog	10	250	500	5
Pig	60	2,400	--	--
Monkey	5	250	500	1.5
Human				
Male	70	--	2,000	rest 7 light work 29 heavy work 43
Female	50	--	2,000	rest 5 light work 16 heavy work 24

(a) Daily food and water consumption and respiratory minute volume vary markedly with animal body weight and age. The standard values presented in this table represent a best estimate for "typical" laboratory animals and are within reasonable limits of the values found in the published literature. In most cases, values have been rounded to two significant figures.

(b) Source: NIOSH (1980).

(c) Source: NIOSH (1980).

(d) Sources: Hamsters -- Hoeltge, Inc.; Humans -- USEPA (1980); Spector (1956); all other animals -- NIOSH (1980).

(e) Source: Rabbit -- Spector (1956); all other animals -- Altman and Dittmer (1972).

(f) Source: Lehman (1959).

TABLE A-2

ACCEPTABLE DAILY INTAKE (ADI)

(Inhalation and Oral Routes)

Chemical	ADI (mg/kg/d)	Organ
Organic Compounds		
Cresols	1.1×10^{-1}	CNS
Ethanol	4.9×10^{-2}	liver
Formaldehyde	1.2×10^{-2}	GI
Methyl ethyl ketone	2.0×10^{-2}	reproductive, CNS
Phenol	1.0×10^{-1}	kidney
Pyridine	9.0×10^{-3}	liver
Styrene	9.5×10^{-1}	liver, kidney
Tetraethyl lead	2.4×10^{-7}	liver, CNS, pancreas endocrine
Toluene	4.3×10^{-1}	CNS
Metals		
Arsenic	4.0×10^{-4}*	CNS
Cadmium	7.5×10^{-4}*	blood
Chromium	2.2×10^{-3}*	liver, kidney
Copper	2.9×10^{-2}	liver, blood
Lead	1.5×10^{-3}	blood
Mercury	5.7×10^{-5}	CNS
Nickel	1.5×10^{-2}*	reproductive
Zinc	2.1×10^{-1}	reproductive, blood

* Oral route only

Unit risks have been developed for a substantial number of carcinogens by EPA's Carcinogen Assessment Group (CAG) and these were adopted for this risk assessment (CAG 1983; USEPA 1982). For other substances that have been designated as either human or animal carcinogens by the Department of Health and Human Services (HHS) or the International Agency for Research on Cancer (IARC), a unit risk score was calculated from carcinogenicity data using the one-hit model. Unless a substance has been designated carcinogenic by EPA, HHS, or IARC, it was not treated as such in this report (IARC, all volumes 1970-1984; HHS-Carcinogen Report References).

Unit risks were calculated using the one-hit model, which takes the following form:

$$\text{Unit risk} = q_1 = \frac{1}{D} \ln \frac{(1 - P_D)}{(1 - P_O)}$$

where P_D = tumor incidence in treated group

P_O = tumor incidence in control group

D = dose in treated group.

A list of the UCR values included in this appendix is presented in Table A-3. The unit cancer risk values are expressed in units of (mg/kg body weight per day)$^{-1}$.

TABLE A-3

UNIT CANCER RISK (UCR)

(Inhalation and Oral Route)

	UCR $(mg/kg/d)^{-1}$
Organic Compounds	
Acrylonitrile	2.4×10^{-1}
Benzene	5.2×10^{-2}
Chloroform	7.5×10^{-2}
Dioxins/Furans	1.6×10^{5}
Formaldehyde	1.12×10^{-2}
Hydrazine	3.9
Methylene Chloride	6.3×10^{-4}
PCB	4.3
Tetrachloroethylene	3.5×10^{-2}
Trichloroethylene	1.9×10^{-2}
1,1,1,-Trichloroethane	1.6×10^{-3}
Metals	
Arsenic	12.7*
Beryllium	2.6
Cadmium	6.6*
Chromium (VI)	41*
Nickel	1.2*

* Inhalation route only

ACUTE SAFE EXPOSURE LIMIT (ASEL)

For several chemicals considered in the risk assessment, only acute exposures were predicted. Acute safe exposure limits (ASEL) were calculated in a manner analagous to that used for chronic exposure ADI (see p. 3), with two significant differences.

First, only data reported for short term exposures, as opposed to chronic exposures, were used to derive the ASEL. Second, the ASEL was based on a NOEL rather than a NOAEL. The short term NOEL was divided by the same safety factors as used for deriving a chronic ADI. In most cases, human data were available for the chemicals of interest in this report, so that a safety factor of 10 would be appropriate. In cases where a NOEL could not be identified, the LOEL was used to estimate the ASEL. In each case of acute exposure where the ASEL was estimated from a LOEL, the LOEL was based on mild effects, requiring the use of either no additional safety factor or a factor of 2.

It should be emphasized that the use of a NOEL for acute exposure studies is considered to be highly conservative and that a no observed adverse effect level (NOAEL) is a more commonly used basis for estimating a safe exposure level. The reason for this is the generally accepted premise (NRC 1979) that some acute effects such as reversible, mild irritation, odor, or sensory motor effects are not adverse if experienced for only a short period. However, to be consistent with the conservative assumptions adopted throughout the risk assessment, any effect (no matter how slight) was considered to be undesirable for the purpose of establishing short term safe exposure levels.

A list of ASEL values for the chemicals reviewed in this report is presented in Table A-4. The more conventional use of NOAELs to establish short term public exposure limits (STPLS) is described in the following section.

TABLE A-4

SHORT TERM LIMITS[a]

Chemical	ASEL[b]	STPL
Organic Compounds		
Methylene chloride	2.5 mg/kg	17 mg/kg
Acid Gases		
Hydrogen chloride	2.6 mg/m^3	4 ppm for 10 min (6 mg/m^3)
Hydrogen cyanide	1.0 mg/m^3	-
Hydrogen fluoride	1.3 mg/m^3	-
Hydrogen sulfide	0.7 mg/m^3	-
Nitrogen oxides	0.2 mg/m^3	-
Sulfur dioxide	0.26 mg/m^3	3 ppm for 10 min (7.8 mg/m^3)

[a] All short term limits based on inhalation exposures

[b] Acute Safe Exposure Levels based on NOEL from short term studies

SHORT TERM PUBLIC EXPOSURE LIMITS (STPLs)

Criteria that can be used to establish short-term exposure limits for air pollutants have been adopted by the National Academy of Sciences (1979). NAS defines short-term exposure as a single exposure, usually lasting 60 minutes or less, but never more than 24 hours. They emphasize that cases where repeated short-term exposure may occur should be differentiated from those in which exposures may never be repeated. Results of human studies should be used to establish these levels in most cases. When human data are not available, the results of animal studies should be used.

The approach used by NAS depends on a substance-by-substance evaluation of each chemical. Exposure limits for compounds, for which reliable human and animal data predict little chance of irreversible effects from a single exposure, can be established directly from the human data with no additional safety factor. If the human data are weak, but the animal data strongly suggest irreversible effects are unlikely from a single exposure, then an uncertainty (safety) factor can be applied depending on the quality of the data. When cancer or some other irreversible effects are expected, then several possible approaches can be used. These include:

- Establish an exposure limit based on an appropriate uncertainty factor as would be done for noncarcinogens; or, depending on the nature of the chemical, include specific warnings to particular segments of the population that may be at additional risk, e.g. in the case of pregnant women exposure to a possible teratogen.

- Use quantitative risk assessment to recommend an exposure level that provides a defined level of risk (i.e. 10^{-5}, 10^{-6} etc.).

Using the above criteria, NAS has established three types of short-term exposure limits. These include short-term public limits (STPLS), short-term public emergency limits (SPELs), and emergency exposure limits (EELs). These have been defined by NAS as follows.

- SHORT-TERM PUBLIC LIMITS (STPLs) are those which relate to predictable single exposures, usually lasting 60 min or less, but never more than 24 h. STPLs are limits that, if not exceeded, are expected to be associated with only a remote possibility of a nonincapacitating, reversible effect in an exposed population. They differ from air-quality standards in that the STPLs are intended for infrequent exposures. A nonincapacitating, reversible effect is one that will not cause a period of disability; will not impair one's vision, visibility, judgment, or ability to breath; and will not interfere with one's ability to escape from the site of the exposure.

- SHORT-TERM PUBLIC EMERGENCY LIMITS (SPELs) are those which relate to unpredictable single exposures, usually lasting 60 min or less, but never more than 24 h, and whose occurrence is expected to be rare in the lifetime of any one individual. SPELs reflect an acceptance of the statistical likelihood of the occurrence of a nonincapacitating, reversible effect in an exposed population. They are designed to avoid significant decrements in performance during emergencies and might contain no uncertainty factor. The acceptable level of risk will depend on the specific compound in question and the type of effect produced by the compound. SPELs are concentrations acceptable to the public in emergencies, such as civil defense or defense of neighborhood property in the case of fire.

- EMERGENCY EXPOSURE LIMITS (EELs) differ from SPELs only in that they apply to defined occupational groups, such as military or space personnel, rather than the general public. Because the relevant populations at risk are more narrowly defined than the general public and are generally younger and healthier, an EEL for a specific chemical may differ quantitatively from the SPEL for the same substance. These values will depend on the available data and the collective judgment of the committee recommending the exposure limits.

Although NAS recognizes that emergency exposure limits for the general public (SPELs) and emergency limits (EELs) for defined occupational groups, i.e., military personnel, could be different, they currently believe a single set of emergency exposure limits (EELs) are sufficient to protect both populations. NAS realizes, however, that for population groups which have a more narrow range of susceptibility, the exposure limits might be somewhat higher.

For the purposes of this risk assessment, the STPL was adopted as the most suitable criterion for assessing short term exposures. A listing of STPLs for the pertinent compounds in this appendix was presented in Table A-4.

ABSORPTION DATA

An attempt was made to estimate the amount of absorption that might be expected for each of the chemicals reviewed. Approximate estimates of absorption were derived from review of the secondary literature. Where the absorption factor for a particular chemical was given as a range, the value representing the upper bound of the range was adopted.

For each chemical, the exposure pathway on which the ADI and/or UCR was based, was identified, and used in assigning an absorption factor (see tables A-5 and A-6) for each chemical. This absorption factor was then used in the individual risk assessments. Because ADI or UCR estimates derived from animal studies do not adjust for the degree of absorption, an absorption factor of one was adopted for the exposure pathway if this was the same pathway on which the ADI or UCR was based, regardless of the value which was cited in the secondary literature. If the pathway of human exposure is different than the pathway by which the ADI or UCR was derived, then the literature-based absorption factors were used for risk assessments. If little or no quantitative data were available, 100% absorption was assumed. For example, qualitative statements regarding absorption, e.g. "readily absorbed", "rapid uptake", etc. were considered as indicators of 100% absorption. In the case of the acid gases, absorption was considered to be 100% by the inhalation route, because of their high water solubility and small molecular weights. Tables A-5 and A-6 list the absorption assumed for each chemical by the inhalation and oral routes of exposure.

TABLE A-5
SUMMARY OF INHALATION ABSORPTION DATA

CHEMICAL	PERCENT ABSORPTION	SOURCE	ABSORPTION FACTOR
Organic Compounds			
Acrylonitrile	No data		1
Benzene	46.9%	(NAS, 1976)	1
Chloroform	49-70%	(USEPA 1980 [1980])	0.7
Cresol	No data		1
Dioxins/furans	29% (for dioxins adhering to particles)	(Schaum (USEPA) 1984)	0.29
Ethanol	No data		1
Formaldehyde	95%	(Egle, 1972, in USEPA (OTS) 1981)	1
Hydrazine	"Well absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Methyl ethyl ketone	75%	(Tada et al., 1972, in Clayton, Vol. IIC, 1982)	1
Methylene Chloride	"Rapidly absorbed"	(Van Oettingen et al., 1949 in Clayton and Clayton, Vol. II B, 1981).	1
PCBs	Efficient absorption	(Albro and Fishbein, 1972, in USEPA, 1980)	1
Petroleum distillates	(mineral spirits, Stoddard solvent)		1
Phenol	80%	(Piotrowski, 1971, in USEPA 1980)	0.8
Pyridine	"Absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Styrene	66%	(W.H.O., 1983)	0.66
Tetrachloroethylene	57%	(Ogata et al., 1971, in USEPA, 1980)	0.57
Tetraethyl lead	30±10%	(USEPA, 1977)	0.4
Toluene	36-67%	(Carlsson and Lindquist, 1977 in USEPA, 1982)	0.67
1,1,1-Trichloroethane	"Readily absorbed"	(Stewart, 1968, in NTP, 1983)	1
Trichloroethylene	58-70%	(Soucek and Ulachova, 1960, in NIOSH, 1973)	0.7
Metals			
Arsenic	85%	(USEPA 1980)	1
Beryllium	No data		1
Cadmium	50%	(Clayton & Clayton 1981)	1
Chromium VI	5%	(USEPA 1984)	1
Copper	No data		1
Lead	46%	(USEPA 1984)	0.46
Mercury	80%	(USEPA 1980)	0.80
Nickel	50%	(USEPA 1980)	1
Zinc	No data		1

TABLE A-6

SUMMARY OF ORAL ABSORPTION DATA

CHEMICAL	PERCENT ABSORPTION	SOURCE	ABSORPTION FACTOR
Organic Compounds			
Acrylonitrile	95%	(Young et al. 1977, in USEPA, 1983)	1
Benzene	50%	(Health and Welfare, Canada, 1979)	0.5
Chloroform	10%	(Fray, et al. 1972 in USEPA 1980)	1
Cresol	No data		1
Dioxins/Furans	No data		1
Ethanol	"Rapid"	(Casarett and Doull, 1980)	1
Formaldehyde	"Rapid uptake"	(Malorny et al., in USEPA (OTS) 1981)	1
Hydrazine	"Well absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Methyl ethyl ketone	"readily absorbed"	(Clayton & Clayton Vol II. C.)	1
Methylene Chloride	No data		1
PCBs	92-98.9%	(USEPA, 1980)	1
Petroleum distillates	No data		1
Phenol	"Readily and rapidly"	(USEPA, 1980)	1
Pyridine	"Absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Styrene	No data		1
Tetrachloroethylene	"Poorly absorbed"	(Clayton and Clayton, Vol. IIV, 1981)	1
Tetraethyl lead	Adults: 10% Children: 42%	(Kehoe, 1961; Rabinowitz, 1974; Zeigler, et al., undated, in USEPA, 1977)	1
Toluene	"fairly complete"	(USEPA, 1982)	1
1,1,1-Trichloroethane	"Readily absorbed"	(Stewart, 1968, in NTP, 1983)	1
Trichloroethylene	"Virtually complete	(Daniel, 1962, in USEPA, 1984)	1
Metals			
Arsenic	95%	(USEPA 1981)	1
Beryllium	No data		1
Cadmium	10%	(NRC 1977)	1
Chromium VI	50%	(USEPA 1984)	1
Copper	40%	(USEPA 1980)	1
Lead	15%	(USEPA 1984)	1
Mercury	100%	(USEPA 1980)	1
Nickel	10%	(USEPA 1981)	1
Zinc	80%	(USEPA 1981)	1

A-2 TOXICITY PROFILES FOR ORGANIC COMPOUNDS

ACRYLONITRILE

Laboratory investigations show acrylonitrile to be toxic by all routes of exposure, including inhalation, oral administration, and ingestion. In acute and subchronic studies, acrylonitrile produces adverse effects on the lungs, kidney, spleen, central nervous system, cardiovascular system, and hematologic system in studies with rats, rabbits, and guinea pigs. Acrylonitrile was teratogenic when given by gavage to pregnant rats (USEPA 1980a).

Acrylonitrile is carcinogenic to rats by inhalation and ingestion in drinking water. In a two-year study (Quast et al. 1980 as cited in USEPA 1980a), male and female rats were administered 35, 100, or 300 ppm acrylonitrile via the drinking water. An increased incidence of tumors of the ear canal gland (Zymbal's gland), tongue, gastrointestinal tract, brain, central nervous system, and pituitary, mammary, adrenal, and thyroid glands occurred in the exposed rats, with some tumors appearing in the low-dose animals. In a long-term inhalation study, tumors were reported in rats exposed to 20 and 80 ppm, 6 hours per day, 5 days per week (ACGIH 1981). Acrylonitrile was also mutagenic in short-term assays in *S. typhimurium* and *E. coli* (USEPA 1980a).

Preliminary epidemiologic evidence suggests that acrylonitrile may cause an increased incidence of cancer of the lung, colon, and lymphatic and hematopoietic systems. However, the results from the studies are confounded by exposures to multiple industrial chemicals (USEPA 1980a; ACGIH 1981). Several studies of Russian and Japanese workers have been reported; toxicity attributed to occupational exposure to acrylonitrile includes effects on the cardiovascular, hematopoietic, nervous, and immunologic systems and the liver (USEPA 1980a).

EPA's Carcinogen Assessment Group estimates a unit risk for acrylonitrile of 2.4 x 10^{-1} $(mg/kg/d)^{-1}$ based on carcinogenicity data from oral studies in animals (CAG 1984).

BENZENE

Central nervous system depression is the primary effect of acute benzene exposure in laboratory animals and humans. Inhalation of 2100-4000 ppm benzene by mice, rabbits and rats leads to narcosis and exposure to higher levels of 10 000 ppm or above leads to death often as a result of respiratory paralysis and cardiac dysfunction (Von Oettingen 1940; Leong 1977; Nahum and Hutt 1934; Morvai et al. 1976 as cited in Sandmeyer 1981). The inhalation LC_{50} in the rat has been reported to be 13 700 ppm (Drew and Fats 1974 as cited in Sandmeyer 1981). The oral LD_{50} in the rat ranges from 900 mg/kg for newborns to 4900 mg/kg for old adults (Kimura et al. 1941 as cited in Sandmeyer 1981). Symptoms associated with acute exposures in humans include nausea, giddiness, headache, unconsciousness, convulsions and paralysis (Sandmeyer 1981). Concentrations in excess of 20 000 ppm lead to death if inhaled for 5 or 10 minutes but deaths following more prolonged exposures ranging from 200-1800 ppm have been reported (Von Oettingen 1940 as cited in Sandmeyer 1981).

Chronic exposure to benzene in an occupational setting is causally related to pancytopenia, a reduction in all the major circulating elements in the blood, including erythrocytes, leukocytes, and thrombocytes. In some cases, decreases in only one type of blood element may occur resulting in anemia, leukopenia or thrombocytopenia. Severe cases can progress to aplastic anemia. These types of changes have been reported in settings where benzene vapor ranges around 60-100 ppm (Hardy & Ellis 1948; Wilson 1942 as cited in Cornish 1980).

The most prevalent observation following chronic benzene exposure among laboratory animals is also leukopenia (Cornish 1981). Rats exposed to concentrations around 50-60 ppm for 5-8 h/d for 4-5 d/wk for a total of between 120-756 hours have developed leukopenia (Deichman et al. 1963 as cited in NRC 1980) and, in the case of one experiment, decreased amounts of DNA in white blood cells, decreased myelocytic activity and an increase in relative numbers of red blood cell precursors (Dobushi et al. 1974 as cited in USEPA 1980b). Chronic oral administration of 50-100 mg/kg as well as chronic subcutaneous injection of 800-1320 mg/kg also resulted in leukopenia in rats (Wolf et al. 1956; Braier 1973 as cited in Sandmeyer 1981).

Benzene has occasionally been shown to have toxic effects on reproductive processes and on fetal development. Female rats exposed to 209.7 ppm benzene for 10-15 days prior to mating showed a complete lack of impregnation. The number of offspring among those rats that did become pregnant was inversely proportional to benzene exposure levels in the range from 0.3 to 209.7 ppm (Gofmekler 1968 as cited in USEPA 1980b). No congenital malformations were observed among offspring of mice injected with 3 mg/kg or administered 1 mg/kg by gavage (Nawrot and Staples 1979 as cited in USEPA 1980b). Exposure of 87egnant rats, mice and rabbits to 2200 ppm benzene during the period of organogenesis for 6-7 h/d produced no overtly toxic effects among offspring other than decreased fetal weight and size, variations in the number of sternebrae and ribs and delayed ossification (Green et al. 1977 as cited in USEPA 1980b).

Benzene is recognized as a human carcinogen, based on epidemiological studies and case reports which convincingly demonstrate that exposure to benzene results in an increased risk of developing leukemia. An increased incidence of leukemia has been demonstrated among workers in rotogravure plants and shoe factories in Italy; among Japanese workers exposed to benzene and other chemicals; among rubber industry workers exposed to various solvents including benzene; and among a group of rubber product workers in the United States (Vigliani and Saita 1964; Ishimaru et al. 1971; Andjelkovic et al. 1976, 1977; McMichael et al. 1974, 1975, 1976a,b; Tyroler 1977 all as cited in USEPA 1980b).

Thus far animal studies have not demonstrated a relationship between experimental benzene exposures and leukemia. However, benzene has been associated with other types of cancer in laboratory animals. F344/N rats and B6C3F1 mice administered 0, 50, 100 or 200 mg/kg (male rats) and 0, 25, 50 or 100 mg/kg by gavage (female rats, male and female mice) 5 d/wk for 103 weeks exhibited increased incidence of neoplasms of the following tissues: Zymbal gland, oral cavity, skin, preputial gland, ovary, mammary gland, harderian gland, lung, liver and lymphoid tissue. The incidences of some of these tumors were increased at all dose levels. In addition various non-neoplastic effects on the hematopoietic system, including bone marrow hyperplasia, lymphocytopenia, leukocytopenia and lymphoid depletion were observed (NTP 1984a).

EPA's Carcinogenic Assessment Group has derived a unit risk of 5.2 x 10^{-2} $(mg/kg/d)^{-1}$ for benzene based on human occupational exposure (CAG 1984).

CHLOROFORM

Chloroform is rapidly absorbed through the lungs, gastrointestinal tract, and to some extent, the skin (Torkelson and Rowe 1981). Oral LD_{50} values in mice, rats and dogs are 0.08, 0.3 and 1.0 mg/kg respectively (Kimura et al. 1971, Hill et al. 1975, Klassen and Plaa 1966, as cited in NRC 1977). High concentrations cause anesthesia and narcosis, gastrointestinal upsets, cardiac effects, and liver and kidney injury (Torkelson and Rowe 1981). A dose of 250 mg/kg caused liver and kidney changes in rats (Torkelson et al. 1976 as cited in Torkelson and Rowe 1981).

Chronically, chloroform most often causes effects in the kidney or liver (Torkelson and Rowe 1981). Liver and kidney injury, characterized by lobular granular degeneration and focal necrosis in the liver and cloudy swelling in the kidneys, was noted in rats, rabbits, and guinea pigs exposed by inhalation to 50 ppm chloroform and in dogs inhaling 25 ppm chloroform 7 hours a day, five days a week for six months (Torkelson et al. 1976, as cited in USEPA 1980c).

Chronic exposure to chloroform vapor ranging up to about 240 ppm was reported to lead to complaints of lassitude, flatulence, dyspepsia, dry mouth, thirst, depression, irritability, and frequent "scalding" micturition among workers. Some workers staggered about in the workplace and experienced nausea after exposure. In another group of pharmaceutical workers, enlarged livers were associated with chloroform exposure.

Increased incidences of delayed ossification of skull bones and wavy ribs were noted in fetuses of rats administered 30 ppm chloroform 7 hours per day on days 6 through 15 of gestation. At 100 ppm, fetuses were reported to have increased incidences of several abnormalities including acaudia, imperforate anus, subcutaneous edema, missing ribs, and delayed ossification of sternebrae (Schwetz 1974, as cited in USEPA 1980c). Doses of 316 mg/kg/d and greater caused acute toxic nephrosis and hepatitis and death of dams, as well as fetotoxicity. In rabbits, oral doses of 100 mg/kg/d or higher were toxic to dams and fetuses (Thompson et al. 1974, as cited in USEPA 1980c).

Chloroform has been shown to produce liver tumors in female mice after oral administration of 128 mg/kg for 78 weeks. Kidney epithelial tumors were observed when an average dose level of 100 mg/kg was administered to rats by gavage for 78 weeks (NCI 1976 as cited in NAS 1977). Roe (1976 as cited in Torkelson and Rowe 1981) noted renal tumors in Swiss mice when administered chloroform at 60 mg/kg/d. However, the significance of these tumors is questionable because they were subsequently seen in control animals of the same strain of mice.

The Carcinogenic Assessment Group of EPA estimates a unit risk for chloroform of 7.5×10^{-2} based on carcinogenicity data from oral studies in animals (CAG 1984).

CRESOL

Exposure to cresol results primarily in adverse effects of the central nervous system, although adverse effects of the liver, lungs, kidneys and spleen have been observed (Deichmann and Keplinger 1982). Cresol may be absorbed through the intact skin or through the mucous membranes of the respiratory and gastro-intestinal tracts. Acute exposure may produce muscular weakness, severe depression, collapse and in severe cases, death. Gastro-intestinal disturbances have also been reported by individuals acutely exposed to cresol. Cresol has a corrosive action on tissues, producing burns and dermatitis. Fatal poisoning may result if substantial areas of the skin are wet by cresol and are not washed immediately after the exposure (H.B. Elkins 1958 cited in ACGIH 1980). Experiments designed to investigate the acute effects of cresol in animals have indicated that exposures must be of high concentration to produce adverse effects. Rats acutely exposed to air saturated with cresol vapors suffered no adverse effects (H.F. Smyth Jr. 1956, cited in W.B. Deichmann and M.L. Keplinger 1982). The following oral LD50s in rats, for the various isomers of cresol have been reported: ortho-cresol 1.35 g/kg, meta-cresol 2.02 g/kg, and para-cresol 1.80 g/kg (W.B. Deichmann and M.L. Keplinger 1963, cited in ACGIH 1980).

Chronic exposure to cresol causes damage to the liver, kidney, and skin. Pathological investigations in humans have shown that the pathological effects caused by chronic exposure to cresol are similar to the effects produced by chronic exposure to phenol. These investigations revealed congestion of the liver with pallor and necrosis of the hepatic cells, kidney tubule damage (O. Wandel 1907 cited in Deichmann and Keplinger 1982), and lung damage characterized by hyperemia, emphysema, edema, and bronchopneumonia with petechial hemorrhages in the pleura (E. Kathe 1906 cited in Deichmann and Keplinger 1982). In addition, destruction of the cellular cytoplasm of the gastrointestinal tract was observed in animals following oral administration of the compound (Species unspecificed cited in ACGIH 1980).

The USEPA has derived an ADI of 7.9 mg cresol/day (equivalent to 1.1×10^{-1} mg/kg/d for a 70 kg individual), based on a TLV value (USEPA 1984).

DIBENZODIOXINS AND DIBENZOFURANS

The polychlorinated dioxins and furans comprise a large class of chlorinated aromatic hydrocarbons which includes some of the most toxic synthetic organic compounds that have been tested to date. Within this group, the compound 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) has been the most thoroughly studied and is generally considered to be the most toxic. Several of the furan congeners are also known to be highly toxic, but they have not been thoroughly tested.

2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is one of the most toxic substances known. It produces a delayed biological response in many species and is highly toxic at low doses to mammals, including humans. Many human exposures to TCDD result from occupational exposure to 2,4,5-trichlorophenol (2,4,5-TCP) or 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), in which TCDD is a contaminant. TCDD has been identified as the cause of numerous outbreaks of chloracne in humans. In addition, investigators have reported muscular weakness, loss of appetite and weight, sleep disturbances, hypotension, abdominal pain, liver impairment, peripheral neuropathy, psychopathological changes, hyperlipemia and hypercholesterolemia, and porphyria cutanea tarda among exposed persons (USEPA 1980d).

Damage to the liver and thymus are the predominant effects of subchronic administration of TCDD in animals. Degenerative changes in the liver and thymus, porphyria, altered serum enzyme concentrations, and loss of body weight occur in rats fed TCDD (NAS 1977). Young female rhesus monkeys given a diet containing TCDD for up to nine months showed loss of facial hair and eyelashes, edema, accentuated hair follicles, and dry scaly skin (Allen et al. 1977 as cited in USEPA 1980d). Five of the eight monkeys died from severe pancytopenia.

TCDD is fetotoxic and teratogenic in various animal species. Fetuses from treated rats have shown reduced body weight, a slight increase in intestinal hemorrhage and edema, and an increase in fetal kidney malformations. In addition, a reduction in fetal number and an increase in fetal deaths have been noted. Increased incidences of cleft palate and kidney abnormalities have occurred in the offspring of treated mice (USEPA 1980d). Gastrointestinal hemorrhage has been observed in the fetuses of treated hamsters (NRC 1977).

TCDD is a potent carcinogen. Ingestion by rats of 2.2 ppb or 0.1 μg/kg/d for lifetime induced squamous cancer of the respiratory tract and oral cavity in males and females and liver cancer in females only. In rats fed diets containing 0.001 to 1000 μg TCDD/kg diet, an increased incidence of liver tumors developed in groups receiving TCDD at levels of 0.005 to 5 μg/kg of diet; animals in the higher dose groups died between the second and fourth weeks of treatment (USEPA 1980d).

EPA's Carcinogen Assessment Group (CAG) estimates a unit risk of 1.6 x 10^5 $(mg/kg/d)^{-1}$ based on data from oral carcinogenicity studies in animals (CAG 1984). The Ontario Ministry of the Environment has also published an ADI for dioxins of 10 pg/kg which is based on use of a safety factor (OMOE 1985).

We will not use this ADI because it was calculated assuming dioxins are carcinogens for which a threshold exists. At the present time there is not enough data to come to such a conclusion.

The most toxic of the furan compounds are certain polychlorinated dibenzofuran (PCDF) congeners. The toxicity seems to depend on the number and position of the chlorine substituents of a congener and is highest for the tetra-, penta-, and hexachloro- compounds. The most toxic of the PCDFs includes those compounds fully chlorinated at the lateral positions, e.g., 2,3,7,8-tetrachlorodibenzofuran and similar congeners (Buser 1985).

Little is known about the toxic effects of PCDFs in man because there are no recorded cases in which individuals have been exposed to pure mixtures of PCDFs. In the Yusho and Yu-Cheng outbreaks, resulting from widespread ingestion of contaminated rice-oil in Japan and Taiwan respectively, individuals ingested a mixture of polychlorinated biphenyls (PCBs), PCDFs, and polychlorinated quaterphenyls (PCQs). A number of investigators feel that the lingering toxic effects seen in these individuals are primarily due to the TCDF fraction which has been shown to be the most toxic in laboratory studies (Masuda et al. 1985; Miyata et al. 1985; Kashimoto et al. 1985). The authors also support this contention by noting the relatively mild and short-termed symptoms that have been reported in workers exposed to relatively pure mixtures of PCBs. Symptoms most frequently reported in Yusho and Yu-Cheng cases were dermatological effects, including chloracne and pigmentation of nails, skin, and mucous membranes; ocular effects, including eye discharge, swelling of upper eylid, and transient visual disturbance; hepatic enzyme induction, and a number of subjective complaints including headaches, numbness of the limbs, and weakness (Reggiani and Bruppacher 1985). A number of these symptoms were also seen in babies exposed in utero or as a result of being nursed with contaminated milk.

The toxic effects noted in toxicity studies of PCDF congeners has revealed a toxicity pattern which is almost identical to that resulting from exposure to other halogenated aromatic hydrocarbons, including TCDD. It appears that these compounds act by a common mechanism (Poland et al. 1983).

In a study in which rhesus macaques were dosed with food containing PCB mixtures, TCDD, or 2,3,7,8-tetrachlorodibenzofuran (TCDF), the same clinical toxic manifestations and histopathologic lesions were observed in the animals, although the potencies of the toxicants covered a wide range (McNulty 1985). Monkeys fed 5 μg/kg/d TCDF in the diet showed atrophy of squamous metaplasia of the sebaceous glands, involution of the thymus, and bone marrow hypolasia. The signs of acute toxicity of TCDF occurred at approximately ten times the dose at which these signs were observed for TCDD.

Different animal species appear to differ greatly in their sensitivity to TCDFs. Guinea pigs are the most sensitive of the animals tested, with an oral LD_{50} of 5-10 μg/kg being reported for this species (Moore 1979). Luster et al. (1979) reported on a study in which guinea pigs were given 2,3,7,8-TCDF by gavage at doses of 0.05 , 0.17 , 0.5 or 1.0 μg/kg once weekly for six weeks. At the two highest doses, thymus/body weight ratios were suppressed and the animals showed suppression of cell-mediated immune function. The authors reported a 30% mortality in the guinea pigs in the highest dosage group.

TCDFs are also potent inducers of the liver microsomal enzyme system. In one study, the ED_{50} (the dose which results in a response in approximately 50% of the population) for the induction of the enzyme aryl hydrocarbon hydroxylase (AHH) in the rat was found to be approximately 0.5 μg/kg/d for five days (Goldstein, 1977).

There is little published information on the reproductive and teratogenic effects of well-characterized PCDFs. It is reported that the treatment of pregnant mice with doses of 10-100μg 2,3,7,8-TCDF/kg on gestation days 10-13 resulted in a dose-related increases in cleft palates and hydronephrotic kidneys in the fetuses (Weber, H. et al. 1984 as cited in Van Den Berg et al., 1985).

No information was identified concerning the carcinogenicity of PCDFs. The structural similarity and parallel toxicity which some of the congeners show with chlorinated dibenzodioxins (i.e., TCDD) gives reason to expect that certain of these (e.g., 2,3,7,8-TCDF) will probably also be identified as potent carcinogens upon testing. For this reason, PCDFs were assumed to be potent carcinogens similar to dioxins and were assigned the unit risk of 1.6×10^5 $(mg/kg/d)^{-1}$ which, as previously described, was calculated for TCDD by EPA's Carcinogen Assessment Group (CAG 1984).

ETHANOL

The acute toxicity of ethanol is low for both animals and man. The LC_{50} in rats is about 13 000 ppm for 22 hours (ACGIH 1981). The symptoms of intoxication are slight irritation of mucous membranes, ataxia, incoordination, and drowsiness. The narcotic dose for rats is 19 260 ppm for 2 hours (ACGIH 1981). Ethanol is not appreciably irritating to the skin, but has caused mild to severe irritation to rabbit eyes depending on the concentration used in testing (Rowe and McCollister 1982). Concentrations of 250 to 1064 ppm for occupational exposure to ethanol vapor have been suggested as nonirritating to the eye and upper respiratory tract (ACGIH 1981). Teratogenic effects including increased postimplantation death, retardation of fetal development, and minor skeletal variants have been reported in mice and rats (NIOSH 1983).

Prolonged exposure to ethanol has resulted in fatty changes in the myocardium and liver. Rats fed a diet in which 33% of the caloric intake was ethanol for up to 14 weeks developed fatty livers (Rowe and McCollister 1982). Rats, guinea pigs, rabbits and beagle dogs exposed continuously to vapors containing <86 mg/m^3 ethanol for 90 days exhibited no effects as measured by behavior, mortality, hematologic effects, and gross histopathology (Coon et al. 1970 as cited in Rowe and McCollister 1982). Exposure of rabbits to air saturated with alcoholic vapors for 25 to 365 days induced cirrhosis of the liver (Mertens 1896 as cited in Rowe and McCollister 1982). There is no information regarding the carcinogenicity of ethanol.

Based on the 90 day study in rats (Coon el al. 1970 as cited in Rowe and McCollister 1982) and applying safety factors of 100 for extrapolation from animals to sensitive humans and 10 for extrapolation from subchronic to chronic effects, an ADI of 4.9×10^{-2} mg/kg/d is estimated.

FORMALDEHYDE

In humans, adverse health effects may occur after exposure to formaldehyde by inhalation, ingestion, or skin contact. Inhalation of formaldehyde causes eye, skin, and respiratory tract irritation. Effects including eye irritation, lacrimation, ocular damage, itching of the eyes, burning of the eyes and nose, sneezing, coughing, dry and sore throat, pricking irritation of the mucous membranes, upper respiratory tract irritation, primary skin irritation, allergic dermatitis, disturbed sleep, headaches, and unusual thirst developed in workers exposed to formaldehyde vapor (ACGIH 1981). Ingestion of formaldehyde causes headaches, upper gastrointestinal pain, allergic reactions, corrosion of the gastrointestinal and respiratory tracts, systemic damage, and death. Systemic poisoning by ingestion is uncommon because the irritant effects of formaldehyde make ingestion unlikely. Direct contact with aqueous formaldehyde causes skin irritation and ocular damage. Formaldehyde also causes skin reactions in some sensitized people using cosmetic formulations containing only 0.01 percent formaldehyde (NRC 1981).

Formaldehyde is irritating to the eyes, skin, and respiratory tract of laboratory animals. Formaldehyde is not teratogenic in animals by ingestion or inhalation; however, oral doses of 185 mg/kg/d formaldehyde (containing 12-15% methanol as a preservative) on days 6 to 15 of gestation caused death in 22 of 34 pregnant mice (Marks et al. 1978 as cited in NRC 1981). No reproductive effects have been observed to occur in male rats exposed to formaldehyde through drinking water or by inhalation, or in dogs exposed to formaldehyde in the diet. Formaldehyde is mutagenic in several test systems (NRC 1981).

In subchronic inhalation studies in rats, guinea pigs, rabbits, dogs, and monkeys, formaldehyde caused lung, heart, and kidney inflammation; decreased liver weight; eye and respiratory tract inflammation; decreased weight gain; and decreased food consumption.

In a chronic inhalation study in rats and mice, sponsored by Chemical Industry Institute of Toxicology (CIIT), formaldehyde caused squamous cell carcinoma of the nasal cavity in rats exposed to 15 ppm, 6 hours/day, 5 days/week for 18 months. At the Formaldehyde Symposium held in Raleigh, N.C., in November 1980, CIIT reported that nasal cancer occurred in rats exposed to 6 ppm for 24 months and in mice exposed to 15 ppm for 24 months (CIIT 1980, as reported in NRC 1981).

The estimated dose-response curve for formaldehyde is very steep. For this reason, the UCR based on the upper 95% confidence limit derived over a large range of exposure concentrations yields extremely conservative and unreliable risk estimates. The UCR used in this analysis was based on the upper 95% confidence limit of the dose-response curve limited to the range of doses of concern estimated in the exposure scenarios. The UCR calculated in this manner is 1.12×10^{-2} $(mg/kg/d)^{-1}$ and still should be considered to be a very conservative estimate. For example, the risk calculated using the above UCR for a theoretical exposure of 3.2×10^{-5} ppm over 30 years of a 70 year lifetime is 2.6×10^{-7}. In contrast, the maximum likelihood estimate of risk for the same scenario is 8.1×10^{-22}, a difference of 15 orders of magnitude. The UCR based on the 95% upper confidence limit was used in the analysis, despite the discrepancies involved, in order to be consistent with other UCRs used in the analysis.

Because there is no evidence that formaldehyde is carcinogenic by ingestion, a toxicity reference dose was estimated for noncarcinogenic effects of formaldehyde. Dubrenil et al. (1976 as cited in NRC 1981) exposed rats continuously to 1.6 ppm formaldehyde for 45 to 90 days. At this dose level, hair discoloration was evident. At a higher dose level weight gain was reduced. Applying a safety factor of 100 to the no adverse effect dose of 1.15 mg/kg/d yields an estimated ADI of 1.2×10^{-2}mg/kg/d.

HYDRAZINE

Hydrazine is a strong skin and mucous membrane irritant, convulsant, and has hepatotoxic and mild hemolytic properties (Reinhardt and Brittelli 1981). Exposure to high concentrations of the vapor results in eye and respiratory tract irritation, lung congestion, bronchitis and pulmonary edema. In rats, the oral LD_{50} is 60 mg/kg and the inhalation LD_{50} for 4 hour exposure is 570 ppm. (Reinhardt and Brittelli 1981). Hydrazine is rapidly absorbed through intact skin, and also through the lungs and gastrointestinal tract. (IARC 1974a).

Hydrazine has been shown to be carcinogenic in mice and rats after oral administration. The major target organ is the lung, although hepatocarcinomas have also been noted (IARC 1974a). In mice fed 21 mg hydrazine sulfate for up to 78 weeks, the incidence of pulmonary tumors was 54% in males and 32% in females, compared with control incidences of 24% and 4% in males and females, respectively (Biancifiori 1970 as cited in IARC 1974a). In rats administered 18 mg hydrazine by stomach tube for 68 weeks, incidences of pulmonary tumors were 3/14 and 5/18 in males and females, respectively (Severi and Biancifiori 1968 as cited in IARC 1974a). In this study, no tumors were found in the untreated animals.

Although it has not been extensively studied, there is no epidemiological evidence that hydrazine is carcinogenic in humans. Roe (1978 as cited in ACGIH 1981) reported no increase in tumor incidence in workers from a hydrazine manufacturing plant in operation from 1945 to 1970. The Carcinogen Assessment Group of EPA has derived a unit risk of 3.9 $(mg/kg/d)^{-1}$ for hydrazine based on oral animal studies (CAG 1984).

METHYL ETHYL KETONE

Acute exposure of humans to methyl ethyl ketone causes irritation of the eyes, nose, and throat (K.W. Nelson et al. 1943 cited in W. J. Krasavage et al. 1982). Exposure to higher concentrations of methyl ethyl ketone (300-500 ppm) also produces nausea and headache (H.B. Elkins 1959 cited in Krasavage et al 1982). In addition, methyl ethyl ketone causes irritation and corneal damage when splashed in the eyes (Clayton and Clayton 1982). In animals, acute exposure to methyl ethyl ketone causes irritation of the eyes and nose and may also cause narcosis or death. Exposure of rats to 4000 ppm for 2 hours produced death in 4/6 animals exposed (H.F. Smyth Jr. 1956 cited in Krasavage et al 1982). Exposure of guinea pigs to 3000 ppm, however, produced no observable adverse effects. The following oral LD50s for rats have been reported 3.3, 3.4 and 3.6 g/kg (E.T. Kimura et al 1971 and H.F.Smyth 1954, cited in Krasavage et al 1982).

Workers chronically exposed to methyl ethyl ketone by inhalation or by skin contact developed numbness in their upper extremities (A.R Smyth and N.R. Mayers 1944 cited in Krasavage et al 1982). In addition, prolonged dermal contact may defat the skin and cause dermatitis (K.E. Malten et at 1968 cited in Krasavage et al 1982). Chronic exposure of animals, by various routes of exposure, does not appear to produce adverse central nervous system effects. Cats and dogs receiving subcutaneous injections of methyl ethyl ketone 5 days per week for 8.5 months showed no signs of nervous system damage (P. S. Spencer and H. H. Schaumburg 1976, cited in Krasavage et al. 1982). Rats receiving intraperitoneal injections of the compound for 35 weeks suffered no neuropathological changes (Unpublished data, Eastman Kodak Company cited in Krasavage et al. 1982). Finally, there was no evidence of neurotoxicity in rats following exposure by inhalation to 4740 ppm for 4 weeks or to 2150 ppm for 6 weeks (P.V. DeJesus et al. 1977, cited in Krasavage et al. 1982).

In a study of the teratogenic effects of methyl ethyl ketone, pregnant rats were exposed to 1000 ppm or 3000 ppm methyl ethyl ketone for 7 hours per day on days 6 through 15 of gestation. No maternal toxicity was observed at either dose level. Litters of rats exposed to 1000 ppm exhibited an increased incidence of skeletal abnormalities; litters exposed to the higher dose level had an increased incidence of sternebral variations, and soft tissue alterations (Schwetz et al. 1974 cited in Krasavage et al. 1982). A repeat of this study indicated that neither dose level produced teratogenic or embryotoxic effects. The slight increased incidence of fetotoxic effects observed in the first study was also noted in the second study (M. M. Deacon et al 1981 cited in Krasavage et al 1982).

The USEPA has derived an ADI of 1.40 mg methyl ethyl ketone per day (equivalent to 2.0×10^{-2} mg/kg/d for a 70kg individual) based on a NOEL obtained in a subchronic inhalation exposure of rats (USEPA 1984).

METHYLENE CHLORIDE

The central nervous system appears to be the primary target organ for methylene chloride. In humans accidental inhalation of high concentrations of methylene chloride of the order of about 20 000 ppm leads to central nervous system (CNS) depression, ultimately resulting in narcosis and perhaps death (Moskowitz and Shapiro 1952 as cited in IPCS 1984). Signs of CNS depression, narcosis, irritation of the eyes and respiratory tract, and lung edema have been reported following exposure to smaller concentrations of methylene chloride vapor via the use of paint removers, and have been associated with corresponding increases in carboxyhemoglobin levels which are indicative of methylene chloride metabolism. Exposure to methylene chloride in a poorly ventilated room has also been associated with the occurrence of myocardial infarction (IPCS 1984).

Exposures of mice to 5000 ppm methylene chloride continuously for 7 days had little permanent effect. Subjects responded to exposure with excitement and increased activity which was followed by decreased activity and "hunched" posture, but by later in the exposure period the mice were virtually normal (Weinstein et al. 1972 as cited in USEPA 1985). Exposures of mice to 1000 ppm continuously for 14 weeks decreased activity levels (Thomas et al. 1972 as cited in USEPA 1985).

CHRONIC TOXICITY

Chronic methylene chloride exposure has been associated with hepatotoxic and nephrotoxic effects. Inhalation exposure to 100 ppm methylene chloride for 100 days resulted in slight cytoplasmic vacuolization in the liver with positive fat stains, and tubular degeneration in the kidneys of rats and mice (Hann et al. 1972 as cited in USEPA 1985). Similar changes in the liver were found in dogs and monkeys exposed for 4 weeks to 1000 ppm methylene chloride. Dogs, cats and guinea pigs have exhibited liver injury following exposures in the range of 7 200 to 10 000 ppm for periods of from 4 to 75 weeks (Heppel et al. 1944 as cited in USEPA 1985; Lehman and Flury 1943 as cited in ACGIH 1981). Repeated methylene chloride exposure to 500 ppm to 5000 ppm has also been shown to increase the metabolic activity of rat liver microsomes (Norpoth et al. 1974 as cited in USEPA 1985).

Exposure of rats and mice to 1250 ppm methylene chloride 7 h/d during days 6-15 of gestation resulted in an increased incidence of dilated renal pelvis among rat pups and an increased incidence of extra sternebrae among mice pups (Schwetz et al. 1975 as cited in USEPA 1985). Exposure of pregnant rats to 4500 ppm methylene chloride 6 h/d, 7 d/wk during pregnancy resulted in decreased body weights among the fetuses (Hardin and Manson 1980 as cited in USEPA 1985). These offspring also showed retarded behavioral habituation to novel environments (Bornschein et al. 1980 as cited in USEPA 1985).

Methylene chloride is considered to be an animal carcinogen. A dose-related increase in incidence of salivary gland region sarcomas was observed in male rats following 2 years of inhalation exposure to 1500 ppm or 3500 ppm methylene chloride for 6 h/d 5 days per week (Dow Chemical Co. 1980 as cited in USEPA 1985). Male mice receiving about 235 mg/kg/d methylene chloride in the drinking water for 24 months had a very slightly increased incidence of hepatocellular adenomas and/or carcinomas (National Coffee Assoc. 1983 as cited in USEPA 1985). In a National Toxicology Program sponsored investigation, 500 or 1000 mg/kg/d methylene chloride was administered by gavage to both male and female Fischer 344 rats and B6C3F$_1$ mice for 2 years. Increased incidences of hepatic neoplastic nodules and adrenal cortical adenomas were observed in both rat sexes, and an increased incidence of pancreatic acinar-cell adenomas was found in males. Hepatocellular carcinomas were observed in both sexes of treated mice (NTP 1982 as cited in NRC 1983). These results however are undergoing re-evaluation as the National Toxicology Program Technical Review Subcommittee concluded that the maximum tolerated dose may have been exceeded in these studies (NRC 1983). An additional National Toxicology Program sponsored bioassay exposing Fischer 344/N rats and B6C3F1 mice to 0, 1000, 2000 or 4000 ppm is currently underway (NTP 1984b).

UCR

EPA's Carcinogen Assessment Group has estimated a unit risk of 6.3×10^{-4} $(mg/kg/d)^{-1}$ methylene chloride, based on carcinogenicity data from oral animal studies (CAG 1984).

ACUTE TOXICITY

The acute toxic effects of methylene chloride on humans include decreased psychomotor performance (Winneke 1974, as cited in USEPA 1980), central nervous system dysfunction and irritation of the mucous membranes (eyes, respiratory tract, and skin) (NAS 1978, as cited in USEPA 1980). Mild poisoning produces somnolence, lassitude, anorexia, and mild lightheadedness. Fatal poisonings have resulted from cardiac injury and heart failure (NAS 1978, as cited in USEPA 1980). Upon metabolism, methylene chloride will form carbon monoxide, which will increase carboxyhemoglobin levels in the blood.

Several investigators have noted the potential risks of elevated carboxyhemoglobin levels to individuals with pre-existing cardiovascular stress (e.g., angina and myocardial ischemia) (NAS, 1980).

Virtually all experimental acute exposure studies of methylene chloride using humans have studied neural or behavioral dependent variables (USEPA, 1983). Stewart et al. (1972b as cited in USEPA, 1983) reported that subjects had symptoms of "lightheadedness" and had difficulty with speech articulation at methylene chloride levels higher than 3012 mg/m^3 for more than one hour. The investigators also reported anecdotal data about increased amplitude in certain visual evoked potentials at about the same exposure levels; however,no statistical analyses were possible because of the small number of subjects (three to eight individuals). In a later study (Stewart el al., 1973, as cited in USEPA, 1983) they reported that 173, 347 or 867 mg/m^3 methylene chloride had no effect on the Romberg equilibrium test, vigilance, coordination or arithmetic skills.

Winneke (1974) and Winneke and Fodor (1976) (both as cited in USEPA, 1983) reported that the central nervous system was impaired during methylene chloride exposure. These investigators exposed female human subjects to 0, 1041, 1730 and 2776 mg/m^3 methylene chloride for up to three hours. They measured performance on a series of hand-eye coordination tasks. They also measured the critical flicker fusion (CFF) frequency and performance on an auditory signal detection task. Concentration related decrements in signal detection, beginning at 1041 mg/m^3 were observed. Many motor decrements were observed at 2776 mg/m^3, the only concentration at which these tests were conducted. Fodor and Winneke (1971, as cited in USEPA, 1983) reported a similar study in which exposure to 1044 and 2776 mg/m^3 resulted in both decreased CFF frequency and impaired signal detection behavior; however, the two methylene chloride levels did not produce effects significantly different from each other.

Putz et al. (1976, as cited in USEPA, 1983) exposed six men and six women (ages 18 to 40 years) to 700 mg/m^3 of methylene chloride for four hours. Eye and hand coordination at the end of the exposure period were depressed as compared with controls. Peak alveolar carbon monoxide after the four hours was 60 mg/m^3 and carboxyhemoglobin was 5.1 percent. These levels are similar to the levels observed for four hour exposures to carbon monoxide, causing the authors to conclude that carbon monoxide may have been primarily responsible for the decreases in performance after exposure. (Other experiments have shown that methylene chloride is more toxic than carbon monoxide (NAS, 1980; USEPA, 1983)).

Gamberale et al. (1975, as cited in USEPA, 1983) studied the effect of methylene chloride exposure on psychomotor and cognitive performance in 14 men, ages 20 to 30 years. The men were exposed for successive 30 minute intervals of 870, 1740, 2600 and 3470 mg/m^3 (half the men were exposed to increasing levels, half to decreasing levels). Total exposure time was two hours. During the exposure, no statistically significant impairment in any measured performance was observed, although reaction time appeared to be affected.

Stewart et al. (1972a, b, as cited in USEPA, 1983) reported no effects of exposure to 1730 or 3460 mg/m^3 methylene chloride for two hours on various hematological, hepatic or renal measures. Continuous electrocardiographic monitoring during one to two exposures (duration unspecified) to 3460 mg/m^3 did not reveal arrhythmias. Gamberale et al. (1975, as cited in USEPA, 1983) reported that methylene chloride levels up to 3460 mg/m^3 produced no alterations in heart rate (duration of exposure unspecified).

Stewart and Dodd (1964, as cited in USEPA, 1983) studied groups of three to five men and women (25 to 62 years old) who immersed their thumbs in various chlorinated aliphatic solvents. Within the first 2 minutes of immersion in methylene chloride, all subjects reported an intense burning sensation on the dorsal surface of their thumbs. Within 10 minutes, a feeling of coldness or numbness, alternating with the burning sensation, was reported; the slightest movements of the thumb triggered waves of searing and excruciating pain. Following removal from the solvent, their thumbs were described as "numb and cold" or "asleep." Within one hour, both the erythema and paresthesia subsided.

Studies of human blood in vitro from individuals homozygous for sickle cell anemia (Matthews et al., 1977, 1978, as cited in USEPA, 1983) have shown that methylene chloride caused hemolysis, preferentially of sickle and other abnormal cell types. These reports suggest that acute exposure to relatively high concentrations of methylene chloride may result in anemia, particularly in individuals who are predisposed to hemolytic anemias.

In addition to the experimental studies discussed above, human toxicity data are available from reports of accidental exposure to methylene chloride. The lowest recorded lethal dose for humans is 500 mg/kg (NIOSH, 1978, as cited in USEPA, 1981). However, quantitative data on exposure levels is usually not available in these cases. Human case studies involving myocardial infarction have been reported from methylene chloride exposure and have included fatalities. For example, methylene chloride was a probable factor in the death of a worker who became unconscious, fell in a vat containing methylene chloride, and died after suffering myocardial infarctions (Kuzelova et al., 1975, as cited in USEPA, 1983). However, Torkelson and Rowe (1981) report that experience with use of methylene chloride has generally been favorable. According to these authors, six reports of death have been found and these deaths were the result of gross over exposure. They state that, most commonly, recovery even following exposure to anesthetic concentrations, has been rapid. Non-fatal exposures have caused electrocardiogram changes, similar to those induced by carbon monoxide (USEPA, 1983). A variety of neurotoxic effects have also been reported (NIOSH, 1976). USEPA (1983) states that the case histories of certain affected exposed individuals suggest that the individuals had pre-existing cardiovascular disease.

Hepatotoxicity has not been reported in any human case report, even following fatal exposures. The only evidence of human nephrotoxicity was a finding of congested kidneys following a fatal exposure. Ocular toxicity other than eye irritation and congested conjunctivae has not been reported (USEPA, 1983).

ASEL

Based on the data presented in the toxicity profile, the acute exposure level (ASEL) i.e., the dose below which adverse health effects would not be expected to occur) for acute exposures, was calculated to be 2.5 mg/kg.

This level of 2.45 mg/kg was based on inhalation studies in humans in which very minor auditory effects were reported. Specifically, one study noted dose-related decrements in auditory signal detection tests when women are exposed to 1041 mg/m^3 methylene chloride for three hours. Assuming that an average woman weighs 60 kg and breathes 16 L/min (at light work) the dose to the women in this study was calculated as follows:

$$\text{Dose} = 1041\ \frac{\text{mg}}{\text{m}^3} \times \frac{\text{L m}^3}{1000\ \text{L}} \times \frac{16\ \text{L}}{\text{min}} \times \frac{1}{60\ \text{kg}} \times 180\ \text{min} = \underline{50\ \text{mg/kg}}$$

A safety factor of 10 is appropriate in this case because this level produced minor effects and a factor of 10 should account for the fact that humans vary in susceptability to the minor effects of chemicals. An additional safety factor is also used when estimating a no effect level from a lowest observed effect level. In this case, a safety factor of 2 is appropriate because of the relatively minor effects observed. The acute safe exposure limit based on this study would therefore be 2.5 mg/kg.

STPL

In the case of methylene chloride, the NAS has not established short-term exposure limits. We will therefore apply the criteria established by NAS (1979) to calculate a STPL for the rare occurrence of a methylene chloride spill. Because of the large amount of human data available it can clearly be established that short-term exposure to methylene chloride, at levels of 1000 ppm or less, are not associated with any irreversible effects on the heart, liver, kidney, or blood (Stewart et al 1972a, b). All other reported effects from short-term exposures are nonincapacitating reversible neurological effects (Table A-7). None of these neurological effects would be expected to be so severe that they would impair the ability of individuals to leave an area if needed. Short-term exposure to levels of 868 ppm was only found to cause lightheadedness while levels of 1000 ppm were found to have no effect on psychomotor or cognitive measures. The lowest level found to affect eye-hand coordination was 200 ppm (Puty et al 1976), but 250 ppm was not found to affect vigilance, equilibrium coordination, or arithmetic skills (Stewart et al 1973).

Given the lack of any organ toxicity at 1000 ppm, coupled with the lack of serious psychomotor and cognitive impairment, ENVIRON recommends that an STPL of 200 ppm (706 mg/m^3) for no more than 1 hour be used. This is based on the use of a small (5X) uncertainty factor because of the numerous human studies that have been conducted with methylene chloride.

TABLE A-7

HUMAN EFFECTS REPORTED AFTER SHORT-TERM EXPOSURE TO VARIOUS LEVELS OF METHYLENE CHLORIDE

Finding	Dose, mg/m^3 (ppm)	Citation*
No effect on psychomotor and cognitive measure	3470 (999)	Gamberale et. al., 1975
No effect on hematology hepatic, or renal measures	3460 (996)	Stewart et. al., 1972a,b
"Lightheaded"	3017 (868)	Stewart et. al., 1972b.
Effects on Hand eye coordination	2776 (799)	Winneke and Fodor, 1976
Effects on signal diction and critical flicker fusion	1041 (300)	Winneke; 1971 Winneke and Fodor, 1976
No effect on equilibrium, vigilance, coordination, arithmetic skills	867 (250)	Stewart et. al. 1973
Effect on eye hand coordination	700 (200)	Puty et. al. 1976.

*All as cited in EPA 1983

PETROLEUM DISTILLATES

Petroleum distillates, as defined here, consist of naphtha (rubber solvent), mineral spirits (petroleum spirits, Varsol), and Stoddard solvent. Each component of petroleum distillates is itself a complex mixture of hydrocarbons with toxicity dependent upon the attendant toxicities of the constituents in the mixture. For each component, there is also the potential for synergistic effects. The predominant adverse effects from exposure to petroleum distillates are eye, nose, and throat irritation, dermatitis, and central nervous system depression.

NAPHTHA (rubber solvent)

Naphtha has a boiling range of 45-125°C and consists primarily of hydrocarbons containing five to seven carbons (C_5 - C_7). Fifteen-minute exposures to human volunteers caused slight transitory irritation at a vapor concentration of 430 ppm and eye irritation and headache at 2000 ppm (NIOSH, 1977). In animals, a vapor concentration of 24 200 ppm produced convulsions followed by death after a 4 hour exposure; impairment of motor coordination and eye irritation was observed at 5300 ppm and 9800 ppm, respectively (NIOSH, 1977). In dogs and rats exposed to naphtha up to 480 ppm for 6 hours per day, 5 days per week for 63 days, no adverse effects were detected (ACGIH, 1980). Using the animal NOEL from this study and applying a safety factor of 1000, a toxicity reference dose of 0.20 mg/kg/d is recommended for the ingestion route of administration.

MINERAL SPIRITS (petroleum spirits, Varsol)

Mineral spirits has a boiling range of 150 to 200°C and consists approximately of 85% aliphatic hydrocarbons and 15% aromatic hydrocarbons. Fifteen men exposed to mineral spirits at a concentration of 1440 or 2880 ppm for an unspecified duration experienced nausea and vertigo (dizziness) (NIOSH, 1977). Human subjects exposed to 360, 720, 1080, and 1440 ppm in 4 consecutive 30 minute periods resulted in no impairment in tests for perceptual speed, numerical ability, manual dexterity, reaction time, or short-term memory (NIOSH, 1977). Eight human subjects exposed to mineral spirits at a concentration of 2300 ppm for 50 minutes resulted in a prolongation in reaction time, a probable impairment in short-term memory, but no impairment in the other three tests. Rector et al. (1966, cited in NIOSH, 1977) exposed rats, guinea pigs, rabbits, dogs, and monkeys to mineral spirits continuously for 90 days at concentrations from 65 to 730 ppm. A decline in body weight was detected in guinea pigs and monkeys at 350 and 320 ppm, respectively. A significant number of deaths occurred only in guinea pigs. Upon microscopic examination of surviving animals, no evidence of toxicity was apparent in heart, spleen, kidney, or liver. However, lung congestion, bronchitis, and mixed inflammatory cell infiltration was observed in all species exposed to the highest concentration of mineral spirits tested (730 ppm). Using the monkey LOEL of 320 ppm, a safety factor of 1000, and a severity factor of 2.14 a toxicity reference dose of 0.10 mg/kg/d is recommended for the ingestion route of administration.

STODDARD SOLVENT

Stoddard solvent is generally considered to be one type of mineral spirits. It has a boiling range of 160 to 210°C and is composed primarily of hydrocarbons containing seven to twelve carbons (C_7 - C_{12}). Six human volunteers exposed to Stoddard solvent for 15 minutes experienced no irritation at 24 ppm, slight transitory eye irritation in one of the subjects at 150 ppm and in all six subjects at 470 ppm (NIOSH, 1977). Cases of aplastic anemia as a result of intermittent long-term exposure to Stoddard solvent has been reported (NIOSH, 1977). Fifteen rats exposed to 1400 ppm for 8 hours resulted in eye irritation, slight loss of coordination, a bloody exudate around the nostrils, and one death (NIOSH, 1977). At 800 ppm, similar effects resulted except there was no loss of coordination. At 400 ppm, no adverse effects were detected. In rats and dogs exposed to 0, 84, 190, and 330 ppm for 6 h/d, 5 days per week for 13 weeks, various hematological changes were observed in rats but the changes were not dose-dependent (NIOSH, 1977). Kidney damage was observed in rats exposed to the highest concentration (330 ppm). In another study, rats, guinea pigs, rabbits, dogs, and monkeys exposed continuously to 290 ppm for 90 days resulted only in minor lung congestion and emphysema in guinea pigs (NIOSH, 1977). Based on the animal LOEL of 290 ppm in guinea pigs and applying a safety factor of 1000 and a severity factory of 2.14, a toxicity reference dose of 0.41 mg/kg/d is recommended for the ingestion route of administration.

The toxicity reference doses for each of the components of petroleum distillates are very similar. They are 0.20 mg/kg/d for naphtha, 0.10 mg/kg/d for mineral spirits, and 0.41 mg/kg/d for Stoddard solvent. The lowest of the three, 0.10 mg/kg/d, is selected to represent the toxicity reference dose for petroleum distillates.

ASEL

Humans exposed acutely to 400 ppm of Stoddard solvent showed no adverse effects (Patty, 1981). In one study, humans exposed to 350 ppm of mineral spirits for 8 hours suffered mucous membrane irritation, and in another study, 30 min exposure to 670-1670 ppm caused nausea, vertigo (loss of balance) and some cardiac irregularities (Patty, 1981). Using 670 ppm as the acute LOEL and applying a safety factor of 20 to protect the sensitive segments of the general population against these effects, the recommended acute safe exposure level (ASEL) is 34 ppm.

STPL

Using the same studies described above for setting the ASEL, a STPL of 67 ppm is proposed based on the acute LOEL of 670 ppm and applying a ten-fold safety factor. This should provide an adequate margin of safety to prevent the occurrance of irreversible adverse effects after short-term exposure to petroleum distillates.

PHENOL

Phenol is readily absorbed by humans by all routes of exposure. Following absorption, the compound is rapidly distributed to all tissues. The predominant effects in humans following chronic exposure are digestive disturbances, effects on the central nervous system, and, possibly skin disorders (Deichmann and Keplinger 1981, USEPA 1980).

Prolonged oral exposure in animals can cause damage to the lung, liver, kidneys, heart and genitourinary tract. Chronic inhalation induces respiratory difficulties, lung damage, loss of weight and paralysis (Deichmann and Keplinger 1981). Rats exposed to phenol in their drinking water for 6 months at various levels exhibited no significant effects at concentrations of 5000 ppm or below. At 7000 ppm, depressed growth occurred in the young (Heller and Purcell 1938). Reduced weight gain developed in rats drinking phenol in water for 12 months (USEPA 1980f). Phenol is not a carcinogen when administered alone; however, when applied to the skin of mice after initiation with known carcinogens, phenol appears to promote the development of tumors. Mutagenicity and teratogenicity have not been thoroughly examined (USEPA 1980f).

Rats were administered 50 or 100 mg/kg/d phenol by gavage over a 6-month period (135 doses). After this period, effects on liver and kidney tissue appeared. Slight liver changes and slight to moderate kidney damage were found in rats receiving 100 mg/kg; slight kidney damage occurred in the 50 mg/kg group (Dow Chemical Co. 1976 as reported in USEPA 1980f). The USEPA has derived an ADI for phenol of 0.1 mg/kg/d based on kidney damage in rats (USEPA 1984).

POLYCHLORINATED BIPHENYLS

Polychlorinated biphenyls (PCBs) produce severe toxic effects in man and animals, particularly following repeated exposure by inhalation and ingestion. Major target organs are the skin and liver; however, the gastrointestinal tract and the nervous system are also affected (USEPA 1980e, Deichmann 1981). In human males occupationally exposed to Aroclor 1254, a slight increase in the incidence of cancer, particularly melanoma of the skin, has been reported (Deichmann 1981). This finding however, has not been confirmed.

Female mink fed Aroclor 1254 for 9 months failed to produce offspring (NRC 1977). Rats receiving oral doses of Aroclor 1254 for 18 months showed an increase in liver weight and a reduction in litter survival (NRC 1977). Male Sprague-Dawley rats fed Aroclor 1254 for 52 weeks showed biochemical changes accompanied by morphologic alterations in the liver (NRC 1980). Reproductive effects were noted in rats and chickens when fed Aroclor 1254 (Deichmann 1981). In rabbits, Aroclor 1254 produced teratogenic effects when ingested (Deichmann 1981). The offspring of rhesus monkeys fed PCBs (Aroclor 1248) at 0.5 ppm in the diet on 3 days per week throughout pregnancy and during nursing were underweight at birth, and were hyperactive and showed impaired learning ability when tested at 6 months of age (Bowman et al. 1981). This dietary concentration corresponds to about 0.01 mg/kg/d.

Several rodent studies suggest that some PCBs are carcinogens and may enhance the carcinogenicity of other chemicals (USEPA 1980e). Aroclor 1260 induced a significant increase in the incidence of hepatocellular carcinoma and neoplastic nodules in female rats fed 100 ppm in the diet for 21 months (Kimbrough et al. 1975).

EPA's Carcinogen Assessment Group estimates a unit risk for polychlorinated biphenyls of 4.3 $(mg/kg/d)^{-1}$ based on carcinogenicity data from oral studies in animals (CAG 1984). This value will be used for our risk assessment. The Ontario Ministry of the Environment (OMOE, 1984) also published a cancer risk assessment on PCB's but we will not use this estimate of the UCR for this assessment because the MOE risk assessment leads to safe exposure levels which are at least 50 times larger than obtained using the CAG (1984) UCR. Several factors contribute to the differences in risk estimates between MOE and CAG, which are outlined below:

Source of Difference	MOE Procedure	CAG Procedure
Data set	Hepatocellular carcinoma only from Kimbrough study	Hepatocellular carcinoma or neoplastic nodule from Kimbrough study
Extrapolation model	Linear extrapolation from 99% upper confidence limit on carcinoma incidence	Application of linearized multistage model to carcinoma/neoplastic nodule incidence
Interspecies extrapolation assumption	Rats and humans assumed at equal risk at same dietary concentration of PCB	Rats and humans assumed at equal risk at same daily dose level in mg/m^2 body surface area/day.

These three differences together account for over a 50-fold difference in risk estimates.

PYRIDINE

Human exposure to pyridine is usually by inhalation. Effects of exposure include transient effects on the central nervous system (CNS) and the gastrointestinal tract. Common symptoms of exposure are headache, dizziness or giddiness, nervousness, insomnia, mental dullness, nausea and anorexia. Pyridine is also a photosensitizer (Clayton and Clayton 1981). The oral LD_{50} in rats is 891 mg/kg (NIOSH 1983).

In animal studies, pyridine is not carcinogenic or mutagenic. In testing for teratogenicity, pyridine injected into chicken eggs causes typical muscular hypoplasia of the legs (Clayton and Clayton 1981).

Exposure of rats for 7 hours per day, 5 days per week for 6 months to either 10 or 50 ppm pyridine vapor did not change either the growth rate or mortality when compared with controls. However, increased liver weight to body weight ratios were noted in the treated rats (EOHS 1971). At vapor concentrations of 6 to 12 ppm in occupational exposure studies, mild symptoms of CNS injury have been noted (Teisinger 1948).

Exposure of rats for 7 hours per day, 5 days per week for 6 months to either 10 or 50 ppm pyridine vapor did not change either the growth rate or mortality when compared with controls. However, increased liver weight/body weight ratios were noted in the treated rats (EOHS 1971). An ADI of 9.0×10^{-3} mg/kg/d was estimated using this study by applying safety factors of 100 for inter and intraspecies variation and 5 for the increased liver weight to body weight ratios noted.

STYRENE

The major effect of acute exposure to styrene is eye, skin respiratory system and mucous membrane irritation. Acute exposure of humans to styrene results in similar irritation symptoms in addition to symptoms such as nausea, vomiting and neurological and psychological disturbances (Stewart et al. 1968 and Rogers and Hooper 1956, as cited din Sandmeyer 1981). Orally, styrene has low toxicity; the oral LD_{50} for rats is 5 g/kg. By inhalation, the approximate LC_{50} is 6000 ppm for a 4 h exposure in rats.

A no effect level for chronic exposure to styrene of between 66.7 and 133 mg/kg/d has been reported (Wolf et al. 1956 and Spencer et al 1942, as cited in Sandmeyer 1982). The higher dose resulted in decreased growth and some liver and kidney organ weight deviation. At 1000 mg/kg/day, mortality was increased.

Occupational studies conducted in Russia indicate that additional symptoms, such as anemia, leukopenia, pancreatic changes, and gastric acidity reduction may also be associated with styrene exposure (Bashisov, 1970 and Volkova et al 1970 as cited in Sandmeyer 1981). Some CNS defects have been observed in children whose mother had been exposed to chemicals such as styrene during pregnancy (Sandmeyer 1981). All of these symptoms however are nonspecific and may relate to solvent effects (Sandmeyer 1981).

Styrene has been shown to increase the incidence of lung tumors in offspring of mice given styrene during pregnancy (Ponomarkov and Tomatis 1979, as cited in IARC 1979). Chromosomal aberrations have been noted in workers exposed occupationally to styrene (Meretoja et al. 1977 as cited in IARC 1979). However, it has yet to be shown that styrene is carcinogenic in humans.

The toxicity reference was estimated using the no effect level of 133 mg/kg/d reported by Wolf et al. (1956 as cited in Sandmeyer 1981). A safety factor of 100 to account for intra and interspecies variation was applied to the daily dose of 95 mg/kg/d, resulting in a ADI of 9.5×10^{-1} mg/kg/d.

TETRACHLOROETHYLENE

Several recent literature reviews have been published regarding the toxicity of tetrachloroethylene (NRC 1977, 1980, 1983; USEPA 1980; Torkelson and Rowe 1981 ACGIH 1980 and IARC 1979). The following information has been summarized from these reviews. Acute inhalation exposures among humans are characterized by central nervous system depression and possibly delayed kidney and liver dysfunction and/or pathology. Symptoms of acute exposure to about 100 ppm for two hours include light headedness, burning in the eyes, congestion, and difficulty in motor coordination. Dizziness, headache, unconsciousness and death can occur as the acute exposure level approaches 2000 ppm. Acute exposures of humans are also sometimes followed by elevations in clinical chemistry measures of liver dysfunction and mild hepatitis.

Central nervous system depression follows acute exposure of laboratory animals to tetrachloroethylene as well, along with abnormal weakness, handling intolerance, restlessness, irregular respiration, muscle incoordination and unconciousness. The minimum narcotic concentration in mice is 2950 ppm and the minimum lethal dose is 6000 ppm. Slight increases in liver weights, total lipid and liver enzymes follow short exposures of animals to concentrations in the 1000 to 6000 ppm ranges, and a dose-related fatty infiltration of the liver has been reported following a 4 hour exposure of mice to 200 to 1600 ppm tetrachloroethylene. Oral LD_{50}'s for rats and mice are 2600 to 1300 mg/kg and 8850 mg/kg respectively. A 4 hour LC_{50} for mice of 5200 ppm and an 8 hour LC_{50} for rats of 5040 ppm have been reported.

Prolonged exposure of humans to tetrachloroethylene leads to irritation of the respiratory tract, nausea, headache, sleeplessness, abdominal pains and constipation. Sustained ambient atmospheric concentrations of greater than 200 ppm can lead to narcosis and death. Workers exposed to between 100-400 ppm tetrachloroethylene in dry cleaning plants exhibit central nervous system depression, dysfunction of the autonomic nervous system, slowed motor nerve conduction velocity and alterations in the EEG. Hepatotoxic effects, which include enlarged liver, cirrhosis of the liver, hepatitis, and increases in serum levels of liver enzymes, and nephrotoxic effects which include uremia, proteinuria and increases in serum creatinine, have also occassionally been reported among chronically exposed populations.

Liver effects predominate in animal studies of chronic tetrachloroethylene exposure. Rats exposed to 600 ppm 6 h/d, 5 days per week for 12 months showed reversible toxic effects in the liver; rats exposed to 230 and 470 ppm 8 h/d, 5 days per week for up to 7 months showed pathological effects in both kidney and liver; and, rats exposed to 1600 ppm 7 h/d for 183 days exhibited enlargement of both kidney and liver. Mice exposed to 200 ppm 4 h/d, 6 days per week for 1, 2, 4 or 8 weeks showed fatty degeneration in the liver, but no liver necrosis and no effects on the kidney. Guinea pigs appear to be especially sensitive exhibiting increased kidney weight and increased liver weight with fatty degeneration after 400 ppm 7 h/d for 183 days, and increases in liver weight after 100 ppm for the same amount of time. Mice also show increased liver weights and hepatocellular swelling in the centrilobular region after 11 days of 100 or 250 mg/kg administered orally.

Excess toxic nephropathy is observed in rats exposed to about 500 or 1000 mg/kg by oral gavage 5 days per week for 78 weeks.

Mice exposed to 300 ppm tetrachloroethylene 7 h/d on days 6 through 15 of gestation exhibit decreased fetal body weight, an increased incidence of subcutaneous edema and increased incidence of delayed ossification of skull bones and split sternebrae. Pregnant rats exposed according to the same paradigm showed an increased incidence of resorptions. There was a reduction in the number of pups born alive among rats exposed to 100 ppm tetrachloroethylene 7 h/d on days 7 through 13 or on days 14 through 20 of gestation. Surviving offspring showed initial neurobehavioral deficits which disappeared with age and some changes in neurotransmitter level which persisted at least until 21 days of age.

Tetrachloroethylene (25-100 μmol/egg) injected into the air space of 6 day old developing chick embryos caused a dose related increase in mortality, and those chicks that survived exhibited excess skeletal anomalies.

Tetrachloroethylene is believed to have a low potential for carcinogenicity in mice. B6C3F1 mice exposed by oral gavage to a time weighted average of 536 or 1072 mg/kg (males) and 386 or 772 mg/kg (females) 5 days per week for 78 weeks showed an increased incidence of hepatocellular carcinomas. Rats exposed to 471 or 941 mg/kg (males) and 474 or 949 mg/kg (females) according to the same paradigm showed no carcinomas. However, these rats suffered from a high rate of mortality due to the early development of toxic nephropathy among experimental subjects, rendering interpretation of the negative response inconclusive. Moreover, the positive control, carbon tetrachloride treated rats, showed a poor response in this study.

EPA's Carcinogenic Assessment Group has estimated a unit risk for tetrachloroethylene of 3.5 x 10^{-2} $(mg/kg/d)^{-1}$, based on data from animal oral studies (CAG 1984).

TETRAETHYL LEAD

Exposure of experimental animals to high concentrations of tetraethyl lead (TEL) has resulted in CNS effects such as irritability, hyperactivity, tremors, spasticity, arched backs and postration (Stokinger 1981). The oral LD_{50} in rats is 17 mg/kg, and the one-hour LC_{50} is 0.85 mg/kg (ACGIH 1983).

In chronic exposures in rats, TEL accumulates in the blood, liver and kidney (Stokinger 1981). Histologic changes occur in the liver, pancreas, anal and endocrine systems by oral, inhalation or cutaneous routes of administration (ACGIH 1983). Schepers (1964 as cited in ACGIH 1983) fed rats tetraethyl lead at concentrations of 0.17 mg and 0.0017 mg/kg five times per week for 21 weeks. Histologic changes, but no changes in body weight were observed.

In humans, TEL intoxication is characterized by insomnia, excessive dreaming, emotional instability and increased erratic physical activity (IARC 1973). No studies to assess carcinogenicity of TEL have been reported.

The data reported by Schepers (1964 as cited in ACGIH 1981) were used to estimate a toxicity reference dose. Safety factors of 100 for intra and interspecies variability, 10 for extrapolation from subchronic exposure, and 5 for the histologic changes observed were applied to the average daily dose of 0.0012 mg/kg/d, resulting in an ADI of 2.4×10^{-7}.

TOLUENE

Toluene is readily absorbed by inhalation and ingestion but less well absorbed through the skin. In humans, inhalation exposure can cause central nervous system (CNS) depression. Symptoms of exposure include altered psychomotor performance, irritability, disorientation and unconsciousness (NAS 1980). Some effects of narcosis are evident at around 200 ppm (NAS 1977). Workers exposed to levels of less than 200 ppm complain of headaches, lassitude and nausea; however, no physical effects occur (ACGIH 1981). Women occupationally exposed to toluene report a high incidence of menstrual disorders as well as effects on embryonic and fetal development (NAS 1980).

In laboratory animals, a 4 h LD_{50} of 8000 to 8800 ppm and a minimum 8 hour lethal concentration of 5300 have been reported.

In animals, subtle cytological and clinical changes as well as behavioral changes occur following chronic inhalation exposure (Clayton and Clayton 1981). Dogs exposed 8 h/d, 6 days per week for 6 months to 2000 to 2660 ppm toluene exhibit intoxication, incoordination and hind leg paralysis. Other effects such as thrombocytopenia, leukocytosis, and chromosome damage in bone marrow cells occur in mice and rats following inhalation of low levels of toluene; however, the purity of the toluene used in those latter studies is questionable (USEPA 1980). When applied to the skin of rats, toluene produces hematological changes (NAS 1980). Toluene is not mutagenic or carcinogenic. Teratogenicity occurs in mice (USEPA 1980).

The USEPA has derived an ADI for 4.9×10^{-1} mg/kg/d for toluene based on central nervous system effects (USEPA 1984).

1,1,1-TRICHLOROETHANE

Several recent literature reviews have been published regarding the toxicity of 1,1,1-trichloroethane (IARC 1979; NRC 1980, 1983; USEPA 1980, 1981, ACGIH 1980 and Torkelson and Rowe, 1981). The following information has been summarized from these reviews.

The primary effect of acute 1,1,1-trichloroethane exposure is depression of the central nervous system. Psychophysiological functions are hindered at concentrations of 350 ppm and eye irritation and dizziness occur at 450 ppm. Anesthetic effects occur at concentrations approaching 500 ppm and death is likely to occur at concentrations in excess of 14 000 to 15 000 ppm. Fatalities which have occurred have been attributed to suffocation and/or acute cardiac arrhythmia. Pulmonary congestion and edema, and mild hepatoxic effects which include fatty vacuolation and elevated urinary urobilinogen, have also been associated with acute 1,1,1-trichloroethane inhalation.

Similar anesthetic and cardiac responses have been reported in animals following acute exposures to 1,1,1-trichloroethane. Central nervous system depression rapidly follows exposure of rats to 18000 ppm, which is the 3 hour LC_{50}. Cardiac arrhythmias follow exposure of dogs to 1,1,1-trichloroethane in the range from 5000 to 10 000 ppm. The oral LD_{50} in mice and rats is about 11 000 mg/kg body weight, the i.p. LD_{50} is 5000 mg/kg. Liver damage has been reported in rats only after exposure to nearly lethal doses.

Animals appear to be able to survive repeated exposures to very high levels of 1,1,1-trichloroethane. Rats exposed to 10000 ppm 1,1,1-trichloroethane 7 h/d 5 days per week for 1 to 3 months, showed staggering gait and weakness after 10 minutes and by 3 hours were semiconscious but survivors had completely recovered the following morning. All rats exposed to 5000 ppm 7 h/d 5 days per week survived over 31 exposures.

Repeated inhalation exposures below 1000 ppm appear to have little discernible toxicological effect. Mice exposed continously to 1000 ppm for 14 weeks showed signficant histopathological effects in the liver as well as increased liver weight and triglyceride content. Fatty changes increased liver weight were also observed among guinea pigs exposed to 1000 ppm daily for 90 days. Exposure of rabbits and dogs to 370 ppm daily for 90 days depressed their body weight. Exposure of rats to 800 ppm for 4 weeks increased liver weights but had little other toxic effect.

Rats administered 750 or 1500 mg/kg by gavage and mice administered time weighted averages of 2800 or 5600 mg/kg 5 days per week for 78 weeks leads to diminished body weight gain and decreased survival time, but no specific histopathological changes which can be identified.

1,1,1-trichloroethane does not appear to interfere with reproductive processes or to have teratogenic effects. No effects on average litter size, average number of implantation sites per litter, incidence of fetal resorptions or fetal body measurements were observed among rats or mice exposed to 875 ppm 1,1,1-trichloroethane vapor 7 h/d from days 6 through 15 of gestation. Similarly, no effects on conception and no toxic effects were observed among offspring of rats exposed to 21000 ± 200 ppm 1,1,1-trichloroethane from two weeks before breeding until day 20 of gestation, or among mice exposed to 1000 mg/kg in their drinking water.

A preliminary study by NTP (1983) indicates a compound-related increased incidence of hepatocellular carcinomas. Male and female mice were administered the compound by gavage at doses of 0, 1500 and 3000 mg/kg/d. The combined incidences of hepatocellular adenomas or carcinomas in male mice was 5/49, 7/49 and 12/499 in control, low dose and high dose groups, respectively. The Carcinogen Assessment Group of EPA has estimated a unit risk value for 1,1,1-trichloroethane of 1.6×10^{-3} $(mg/kg/d)^{-1}$ (CAG 1984).

TRICHLOROETHYLENE

Several recent literature reviews have been published regarding the toxicity of trichloroethylene (NRC 1977, 1980, 1983; Torkelson and Rowe 1981, USEPA 1980, ACGIH 1980 and IARC 1979). The following information has been summarized from these reviews.

In humans, acute exposure to trichloroethylene leads primarily to central nervous system depression. Other reported symptoms include visual disturbances, mental confusion, fatigue, tremors, dizziness, nausea and vomiting. High doses also lead to acute renal failure, anuria, uremia and hepatic and cardiovascular damage. Death is frequently attributed to ventricular fibrillation and cardiac arrest. The lowest reported oral lethal dose has been 50 mg/kg. The lowest reported inhaled lethal dose has been 3000 ppm for 10 minutes although concentrations higher than this are often nonfatal.

A low acute toxicity of trichloroethylene is apparent among laboratory animals with the major effect being mild hepatotoxicity. The oral LD_{50} in rats is 4920 mg/kg; in mice is 3200 mg/kg and in dogs is 2800 mg/kg. At near lethal oral doses, mild hepatic dysfunction has been reported in rats. Central nervous system symptoms predominate following high short inhalation exposures although hepatic injury has been reported in rats following a 2 hour exposure to 10000 ppm trichloroethylene. Rats exposed for several hours to 200, 400 to 800, or 1600 ppm trichloroethylene exhibited an increase in spontaneous motor activity, inhibited swimming, and decreased motor activity respectively.

Chronic exposure of laboratory animals to trichloroethylene vapor appears to result in mild hepatotoxic and nephrotoxic effects. Increased liver weight has been observed in rats, guinea pigs and rabbits exposed to 400 ppm trichloroethylene 7 h/d, 5 days per week for 6 months. Increased kidney weights were observed in rats and rabbits if the exposure level was 3000 ppm. Rats exposed to 55 ppm trichloroethylene 8 h/d, 5 days per week for 14 weeks also have increased liver weights, and mice exposed to 1600 ppm 7 to 8 h/d 6 days per week for 8 weeks show a slight accumulation of lipids in the liver.

Histological changes in the brain, especially the cerebellum, have been reported in dogs following exposure to 500 to 3000 ppm trichloroethylene 2 to 8 h/d 5 days per week for a total of up to 162 hours.

Trichloroethylene exhibits slight reproductive toxicity but has apparently no embryotoxic or teratogenic potential. Sperm from mice exposed to 0.3% trichloroethylene vapor 4 h/d for 5 days were reported to have increased abnormalities. No fetal toxicity or teratogenicity was found among offspring of mice or rats exposed to 3000 ppm trichloroethylene 7 h/d on days 6 through 15 of gestation. Anomalies of skeletal and soft tissue indicative of developmental delay were observed among offspring of rats exposed to 1800 ppm 6 h/d from the first through the twentieth day of gestation. Dams showed an increased incidence of displaced right ovary.

The carcinogenic potential of trichloroethylene has been the subject of several investigations. The results of an original investigation showing a dose-related increase in the incidence of hepatocellular carcinomas in mice following oral exposure to trichloroethylene, which had been questioned on methodological grounds, have largely been confirmed. In the original study

B6C3F1 mice exposed by oral gavage to a time weighted average of 2339 or 1 169 mg/kg (males) or 1739 and 869 mg/kg (females) 5 times per week for 78 weeks and sacrificed at 90 weeks showed an increased incidence of hepatocellular carcinomas. Osborne-Mendel rats exposed to 1097 or 549 mg/kg according to the same paradigm and sacrificed at 110 weeks exhibited no increase in tumors. An increased incidence of hepatocellular carcinomas among trichloroethylene exposed B6C3F1 mice was confirmed in a second study in which subjects were exposed to 1000 mg/kg 5 days per week for 103 weeks. Fischer 344 rats exposed to 500 or 1000 mg/kg at the same time failed to show a carcinogenic response.

A modest increase in the incidence of hepatocellular carcinomas has also been observed in B6C3F1 mice, but not Charles River rats, exposed for 2 years, 6 h/d, 5 days per week to 600 ppm trichloroethylene. In another inhalation study, female Han:NMRI mice exposed to 100 or 500 ppm trichloroethylene 6 h/d, 5 days per week for 18 months and sacrificed at 30 months showed an increased incidence of malignant lymphomas. Han:Wist rats and Syrian hamsters exposed according to the same paradigm were unaffected. Results of this last study have been questioned because of a higher than expected incidence of lymphomas in the unexposed mice. Therefore, more inhalation studies involving B6C3F1, Swiss albino, Swiss and ICR/Ha mice and Sprague Dawley rats are under way. An additional gavage study using Osborne-Mendel, Marshall 540, August 28807 and ACI rats is also in progress.

EPA's Carcinogen Assessment Group has estimated a unit risk for trichloroethylene of 1.9×10^{-2} $(mg/kg/d)^{-1}$ based on carcinogenicity data from oral studies in animals (CAG 1984). There is currently no epidemiological evidence which suggests that trichloroethylene is carcinogenic in humans.

A-3 TOXICITY PROFILES FOR METALS

ARSENIC

The toxicity of arsenic compounds varies greatly depending on the chemical form (organic or inorganic) and the oxidation state of arsenic. Trivalent compounds are more toxic than pentavalent compounds.

Long-term inhalation of inorganic arsenic compounds affects the respiratory system in humans (including perforation of the nasal septum). Chronic exposure to arsenic is also associated with skin lesions, peripheral neuropathy, cardiovascular disorders, and peripheral vascular disorders (blackfoot disease) (IARC 1980, USEPA 1980). Smelter workers exposed to arsenic compounds (and possibly other toxic compounds) during pregnancy have had an increased number of infants with low birth weights and increased frequencies of abortion and malformations. Arsenic compounds have also increased the frequency of chromosomal aberrations in humans (IARC 1980, USEPA 1980i).

In several mammalian species, arsenic and arsenate compounds are embryotoxic and/or teratogenic when given orally or by injection (USEPA 1980, IARC 1980). In addition, enzyme systems in a wide variety of species are inhibited by trivalent arsenic compounds (USEPA 1980i). Arsenic compounds induce chromosomal aberrations and morphological changes in mammalian cells.

Inorganic arsenic compounds are classified as human carcinogens by IARC (1980) causing skin cancer in persons exposed to inorganic arsenic through drugs, drinking water, and pesticides and lung cancer in workers exposed to airborne arsenic compounds. In addition, excess incidences of liver cancer, lymphomas, leukemia, myeloma, and colon cancer in various occupationally-exposed groups occurred (IARC 1980, USEPA 1980). The evidence for carcinogenicity in animals is limited. Increased incidences of leukemia and lymphomas occurred in mice given subcutaneous injections of sodium arsenate during pregnancy and lung tumors developed in rats given a calcium arsenate-copper mixture by intratracheal administration (IARC 1980, USEPA 1980).

The EPA's Carcinogen Assessment Group (CAG) has developed a UCR based upon the studies conducted in Taiwan (Tseng et al. 1968; Tseng 1977) in which an increase in skin cancer was associated with the amount of ingestion of arsenic-contaminated drinking water. The EPA Office of Drinking Water established a Maximum Contaminant Level (MCL) and primary drinking water standard under the Safe Drinking Water Act for arsenic at 0.05 mg/L. In addition, the Safe Drinking Water Committee of the NAS indicated that 0.05 mg/L provides a sufficient margin of safety (NRC 1983). If the CAG risk estimates are applied to the MCL, however, a lifetime cancer risk of ca. 2.5 per 100 is projected. Clearly, EPA cannot have accepted a risk of this magnitude (which is of epidemic proportion) in establishing the MCL. Thus EPA does not appear to view the CAG estimates as realistic. At this time there are no other relevant studies upon which to base an oral UCR for arsenic. Consequently an ADI for ingestion was established as described below.

Mizuta et al. (1956 as cited in NRC 1977) observed that human ingestion of 0.04 mg/kg/d of calcium arsenate resulted in facial edema and peripheral neuropathy in persons exposed over a 2 to 3 week period. Applying a 100-fold safety factor to this human LOEL results in an ADI of 4.0×10^{-1} µg/kg/d for the ingestion route.

EPA's Carcinogen Assessment Group developed an estimate of excess lung cancer risk associated with arsenic inhalation in workroom area which translates to a unit cancer risk of approximately 12.7 $(\text{mg/kg/d})^{-1}$ (Federal Register 1983).

BERYLLIUM

Human toxicity from exposure to beryllium is primarily a result of the inhalation of beryllium compounds. Acute toxic effects of beryllium inhalation include nasopharyngitis, tracheobronchitis, and in severe cases chemical pneumomitis, which may lead to pulmonary edema and death (Hammond and Beliles, 1980). Contact dermatitis and other dermatological effects (ulceration and granulomas) have been documented in exposed workers. Chronic beryllium disease primarily affects the respiratory system and results in dyspnoea, cough, and weight loss. The respiratory disease is classified as a granulomatosis and may eventually lead to fibrosis, emphysema, and cor-pulmonale. Hypersensitization appears to play an important role in the disease and might result in the development of beryllium disease in individuals with low exposures.

Beryllium appears to have the potential to cause mutations. Positive mutagenic responses have been reported in studies using cultured mammalian cells. Beryllium compounds have also been found to cause chromosomal aberrations in cultured human lymphocytes.

Positive evidence of carcinogenesis has been reported in some mammalian studies. Carcinogenesis has been successfully induced in rabbits by intravenous or intramedullary injection and also in rats when exposed by inhalation or intratracheal injection. Using the IARC criteria requires that beryllium be placed in the "sufficient evidence" category of animal carcinogens (USEPA, 1984).

The evidence for the carcinogenicity of beryllium and beryllium compounds in humans is inconclusive. Early studies (IARC, 1972, 1980; Bayliss et al., 1971; Bayliss and Lainhart, 1972 as cited in USEPA Review Draft, 1984) did not report any positive evidence of carcinogenicity in humans, but more recent studies indicated an increased risk of lung cancer in beryllium-exposed workers (USEPA Review Draft, 1984). The overall animal and human evidence for the carcinogenicity of beryllium results in its placement in a IARC category for chemicals which are "probably carcinogenic in humans" (USEPA Review Draft, 1984).

EPA's Carcinogen Assessment Group estimates a unit risk for beryllium of 2.60 $(mg/kg/d)^{-1}$ based on carcinogenicity data from studies involving human occupational (inhalation) exposure (USEPA, 1985).

CADMIUM

Human toxicity from exposure to cadmium is well-documented. Acute toxic effects of cadmium inhalation include irritation of the upper respiratory tract, chest pains, nausea and diarrhea, dizziness, and death usually due to massive pulmonary edema. Major toxic effects of cadmium ingestion include nausea, vomiting, salivation, diarrhea, abdominal cramps, and in some cases death (Doull et al. 1980). Following chronic exposure to cadmium the principal target organs are the lungs and kidney. Chronic inhalation of cadmium fumes and dust can lead to an emphysema-like condition with loss of ventilatory capacity, increased residual lung volume, and shortness of breath (Doull et al. 1980). The kidney appears to be the most cadmium-sensitive organ, primarily because of its prediliction for accumulation of cadmium. Renal tubular damage is the most important effect of cadmium exposure, irrespective of the route of exposure. The damage is usually characterized by proteinurea; other signs of renal tubular damage may include a reduced ability to concentrate urine, glycosuria, hypercalcinuria, aminoaciduria, and increased uric acid excretion. Excessive exposure to cadmium through the ingestion of contaminated food and drinking water results in a disease characterized by osteomalacia and renal tubular dysfunction (Itai-itai disease).

The toxic effects of cadmium observed in humans also occur in experimental animals. Rats exposed to a cadmium aerosol for 15 days develop lung inflammation followed by emphysema and fibrosis. Signs similar to those of Itai-itai disease develop experimentally in rats given an excess of cadmium in a calcium deficient diet. In newborn animals, cadmium causes cerebral and cerebellar damage. Cadmium is also toxic to the testes of rats and mice, and causes hyperglycemia and glucose intolerance in animals. Cadmium is teratogenic and fetotoxic in rats, mice, and hamsters (Doull et al. 1980). With regard to mutagenicity, cadmium can cause chromosomal or mitotic aberrations in mammalian cell assays and increase error frequency in DNA synthesis. Mixed results have been obtained for cadmium in microbial mutagenicity assays. Drosophila studies and dominant lethal mutation studies on cadmium were negative (USEPA 1980i).

Several epidemiological studies of workers exposed to cadmium oxide dust provide evidence that cadmium increases the risk of prostatic cancer and respiratory cancer (Stokinger 1981a, USEPA 1980, NAS 1980). IARC (1976) concludes that cadmium and certain cadmium compounds are probably carcinogenic to humans. Animal studies demonstrate that the injection of certain cadmium compounds causes both sarcomas at the site of injection and testicular tumors (Leydig or interstitial cell tumors). Several long-term feeding and inhalation studies have been carried out with cadmium compounds; the induction of tumors by these routes of exposure has not occurred (USEPA 1980j).

EPA's Carcinogen Assessment Group estimates a unit risk for cadmium of 6.6 (mg/kg/d)$^{-1}$, based on carcinogenicity data from studies involving human occupational (inhalation) exposure (CAG 1984).

The National Academy of Sciences has derived a Suggested No-Adverse Level (SNARL) using data from Decker et al. (1958 as cited in NRC 1980). In this study, rats were administered cadmium in drinking water at a concentration of 10 ppm for one year without effect. The rats consumed an average of 30 mL/d in this study. After exposing rats to 50 ppm cadmium in drinking water for 3 months, effects such as anemia were observed. EPA did not consider cadmium a carcinogen by the oral route when it developed its Ambient Water-Quality Criterion (USEPA 1980j). NAS applied a 1000-fold safety factor to the 10 ppm (or 0.75 mg/kg/d) NOEL to derive the chronic SNARL.

Based upon the Decker Study, and applying a 1000-fold safety factor, an ADI of 7.5×10^{-1} µg/kg/d is recommended for the ingestion route of exposure.

CHROMIUM

Chromium compounds can occur in any of three oxidation states, 0, +3, and +6. The biological properties of chromium compounds vary with the oxidation state. Chromium (VI) is the most hazardous form of chromium.

The pulmonary carcinogenicity of chromium, presumably chromium (VI), is well-documented for humans. Increased incidences of lung cancer are associated with occupational exposure to chromium compounds by inhalation; there is no evidence of increased risk of cancer from ingestion (IARC 1980, USEPA 1980k). In addition, inhalation exposure to elevated concentrations of chromium (VI) compounds causes mucosal irritation, ulceration, and perforation in the respiratory system of humans. Dermal exposure results in primary irritation and allergic contact sensitization in relatively high numbers of exposed individuals (USEPA 1980). An increased incidence of chromosomal aberrations occurred in workers exposed to chromium (VI) compounds (IARC 1980, USEPA 1980k).

Administration of Cr (VI) to experimental animals by various routes causes liver damage, kidney damage (confined to the proximal convoluted tubules), and chronic irritation of the respiratory tract. Some embryotoxic and teratogenic effects developed in mice and hamsters given Cr (VI) at very high doses; it is unclear whether these effects are due to maternal toxicity (IARC 1980). Chromium compounds cause mutations, cell transformations, and chromosomal aberrations in numerous prokaryotic and eukaryotic systems, both *in vivo* and *in vitro* (USEPA 1980k, IARC 1980).

The carcinogenic potential of chromium (VI) in animals either by inhalation or ingestion has not been successfully demonstrated. Tumors in experimental animals have been induced only by implantation of pellets containing chromium (VI) compounds or by injection of chromium (VI) compounds (IARC 1980, USEPA 1980).

EPA's Carcinogen Assessment Group (CAG 1984) estimates a unit risk for chromium of 41 $(mg/kg/d)^{-1}$ based on carcinogenicity data from studies of human occupational exposure by inhalation. For noncarcinogenic endpoints, EPA has estimated an ADI of 2.2×10^{-3} mg/kg/d based on an oral study in rats (USEPA 1984).

COPPER

Copper toxicity produces a metallic taste in the mouth, nausea, vomiting, epigastric pain, diarrhea, and depending on the severity, jaundice, hemolysis, hemoglobinuria, hematuria, and oliguria (USEPA 19801). The toxic intake level for inorganic copper for an adult male is greater than 15 mg/day. Data on effects of chronic exposure to copper come from studies of vineyard workers exposed to copper sulfate and lime mist and copper miners exposed to copper sulfide and oxide dusts. In the former instance, some evidence of copper deposition in the lung and cancer of the lung and liver was found. Copper miners apparently do not show any evidence of an increased body burden of copper (NAS 1977).

Chronic toxicity of copper has been studied in animals and a wide variability in sensitivity to copper has been found, e.g., sheep are very sensitive, rats are very resistant. Copper toxicity in sheep and cattle is characterized by excessive hepatic stores, hemoglobinuria, and hemolysis (NAS 1977). Little data exist concerning the carcinogenicity, mutagenicity, and teratogenicity of copper.

In swine and rats the chronic NOEL in the diet was 250 ppm; applying a safety factor of 100 to this figure would result in a daily dose of 150 µg/kg/d (Boyden et al. 1958; Suttle and Mills 1966a, 1966b; Wallace et al. 1960, all as cited in NRC 1977).

The National Academy of Sciences (NAS), however, (1980 as cited in NRC 1980) indicates that 2 to 3 mg/d (equivalent to 28.6 to 42.9 µg/kg/d) is an adequate and safe intake of copper for adults. These levels are set to assure a balance between essential and toxic exposure to copper; any intake within the recommended range is considered adequate and safe. The NAS analyses indicate that the EPA drinking water standard of 1 mg/l (28.6 μg/kg/d) would appear adequate to protect the health of persons from toxicity due to copper in drinking water.

Based upon the lower "safe and adequate" limit for adults (NRC 1980), an ADI of 28.6 μg/kg/d is recommended.

LEAD

There are various physiological levels and exposure ranges at which the effects of lead in man occur. The most important effects are those in the hematological, neurobehavioral, and reproductive systems (NAS 1972).

In the hematopoeitic system, anemia is a clinical feature of lead intoxication, resulting from both increased erythrocyte destruction and decreased hemoglobin synthesis (USEPA 1977). In children, who are more sensitive to this effect than adults, a threshold blood lead level for production of this effect is approximately 4 mg/100 mL. Hemoglobin synthesis is impaired by lead which inhibits the synthesis of the globin moiety and several steps in the synthesis of heme. The step most sensitive to lead in the heme synthetic pathway is that mediated by the enzyme δ-aminolevulinic acid dehydratase (δ-ALAD). Inhibition of δ-ALAD occurs at blood lead levels as low as 10 μg/100 mL. Though the significance to human health of δ-ALAD-inhibition at a blood level of 10 μg/100 mL is open to debate, the increased urinary excretion of δ-aminolevulinic acid that occurs with increasing inhibition of 40 μg/100 mL is accepted as a measure of probable physiological impairment in vivo (USEPA 1977). This threshold level for urinary δ-ALAD excretion appears to apply to both adults and children.

An increase in free erythrocyte protoporphyrin (FEP) occurs at blood lead levels of 15 μg/100 mL in children. Elevation of free erythrocyte protoporphyrin has the same implications of physiological impairment in vivo as urinary δ-ALAD. To the extent that the protoporphyrin elevation is a likely indicator of the impairment of mitochondrial function in erythroid tissue, it may be even more important.

The most severe effects of lead poisoning, acute or chronic encephalopathy and/or death, may occur in some adults at blood levels slightly below 100 μg/100 mL (USEPA 1977). For children, encephalopathy occurs at lower levels down to about 80 μg/100 mL. The blood lead levels at which neurobehavioral deficits occur in otherwise asymptomatic children appear to start at a range of 50 to 60 μg/100 mL.

Placental transfer of lead has been demonstrated both by fetal tissue analysis and comparison of newborn umbilical cord blood lead with maternal blood lead. The prenatal exposure of the fetus to lead, even in the absence of teratogenic effects, adversely affects health. Studies have related fetal lead levels to changes in fetal heme synthesis and to the incidence of premature births. Maternal blood lead levels of approximately 30 μg/100 mL may be associated with a higher incidence of premature delivery and premature membrance rupture, but these observation require confirmation (USEPA 1977).

The effects of lead exposure described for humans also occur in a variety of animal species. In addition, carcinogenicity assays have been performed using several lead-containing compounds. Although data exist on animals which have experienced a carcinogenic effect for certain lead salts, no conclusive evidence exists that inorganic lead or organic lead compounds are carcinogenic (IARC 1980). Unfortunately, a paucity of information exists on the teratogenicity and developmental toxicity of chronic low or moderate lead exposures. Other data suggest, however, that chronic low-level lead exposure may induce postnatal developmental delays in rats.

Based on data on blood lead levels associated with hematologic effects in children (Maximum safe blood lead level for children = 30 μg/100 mL), background lead consumption, and correlations between blood lead levels and oral lead intake, USEPA (1980m) estimates that a water concentration of 50 μg/L is an upper limit of acceptability for lead in water. This is equivalent to intake of 1.5 μg/kg/d (assuming 2 liters per day and a 70 kg adult). This is the ADI recommended for the oral and inhalation routes.

MERCURY

Chronic exposure to inorganic mercury produces symptoms which vary widely among individuals. Symptoms may include fatigue, weakness, loss of appetite, loss of weight, insomnia, indigestion, diarrhea, metallic taste in the mouth, increased salivation, soreness of mouth or throat, gum inflammation, black line on the gums, loosening of teeth, irritability, loss of memory, and tremors of the eyelids, lips, tongue, or fingers. Relatively high exposures produce extreme irritability, excitability, delirium with hallucinations, melancholia, anxiety, or manic depressive psychosis. Generally, chronic exposure to mercury produces gingivitis, excess salivation, increased irritability, and muscle tremors (NIOSH 1977).

The principal effects of chronic mercury exposure in laboratory animals are renal damage and nervous system toxicity. Fitzhugh et al. (1950, as reported in Stokinger 1981a) observed granularity, fibrosis, and enlargement of the kidneys, and hypertrophy and dilation of the proximal convoluted tubules in rats chronically fed mercuric acetate. Peripheral nervous system damage occurs in rats (USEPA 1980n). In rats exposed prenatally to mercury vapor, death occurs within 6 days of birth (Baranski and Szymczyk 1983, as reported in USEPA 1980). Gale and Ferm (1971, as reported in USEPA 1980n) observed teratological effects in "animals" exposed to mercuric chloride. In mice treated with mercuric chloride, reversible inhibition of spermatogonial cells developed (Lee and Dixon 1975, as reported in USEPA 1980).

Druckey et al. (1957, as reported in USEPA 1980n) reports that when metallic mercury is injected intraperitonally into rats, sarcomas develop only in those tissues that are in direct contact with the metal. No other data were found on the potential carcinogenicity of inorganic mercury.

EPA has derived an ADI of 2.8×10^{-4} for mercury. According to USEPA (1980n) it appears that the earliest detected nervous system effects of methyl mercury exposure in man are at blood concentrations of 200-500 ng Hg/mL, which correspond to long-term daily intakes of 200 to 500 mg Hg/70 kg. The 200 µg/d dose corresponds to 2.86 µg/kg/d. In establishing its ambient water quality criterion, EPA applied a 10-fold safety factor to this LOEL for methyl mercury. The EPA drinking water standard (maximum contaminant level) is 2 µg/L, corresponding to a toxicity reference dose of 0.057 µg/kg/d, providing a safety margin of 50 to the LOEL. As all mercury exposure is unlikely to represent methyl mercury, the application of 50-fold safety factor is reasonable and therefore, an ADI of 5.7×10^{-2} µg/kg/d is recommended.

NICKEL

In humans, dermal sensitization is the most common toxic reaction resulting from exposure to nickel and nickel compounds. Dermal exposure may also result in chronic eczema or neurodermatitis. Exposure to nickel and its compounds can lead to irritation of the conjunctiva and the mucous membranes of the respiratory tract (NIOSH 1977). Asthmatic lung disease is documented for nickel plating workers. Cancers of the lung and nasal sinuses occur in nickel refinery workers.

In experimental animals, oral administration of nickel metal is comparatively non-toxic; dogs and cats tolerate up to 12 mg nickel per kg daily for periods up to 200 days without ill effects (Stokinger 1983, as reported in USEPA 1980o). At higher doses acute gastrointestinal irritation with vomiting and diarrhea occurs. By subcutaneous or intravenous injection, gastrointestinal and central nervous system effects, including tremor, chorea-like movements, and paralysis, also arise (Browning 1969).

One researcher found malformations in hamster embryos when the mother was exposed to unidentified nickel compounds administered parenterally at dosages ranging from 0.7 to 10.0 mg/kg (Ferm 1972, as reported in USEPA 1980o). No teratogenic effects occurred when either nickel chloride (16 mg/kg) or nickel subsulfide (80 mg/kg) was administered to rats (Sundermand et al. 1978, as reported in USEPA 1980). Multigenerational reproductive studies have linked oral administration of nickel in the diet to decreases in litter size, and increases in numbers of runts and in neonatal mortality (USEPA 1980o). Male rats given daily oral doses of nickel sulfate at 25 mg/kg were completely sterile after 120 days (Waltschewa et al. 1972, as reported in USEPA 1980o). Nickel chloride was not mutagenic in a host-mediated assay using NMRI-strain mice. In a study of mutation in a streptomycin-dependent E. coli strain, the nickel compounds, $NiCl_2$, $NiSo_4$, and $Ni(No_3)_2$ were not mutagenic (Stokinger 1981).

A significantly increased incidence of cancers of the lungs and nasal cavities occurred in epidemiological studies of workers in nickel smelters and refineries (NRC 1977). The implicated compounds were nickel subsulfide, nickel oxides, nickel carbonyl vapor, and soluble aerosols of nickel sulfate, nitrate, or chloride (NRC 1980). Tumors developed in rats, mice, guinea pigs, and hamsters administered nickel compounds by inhalation, and by the subcutaneous, intramuscular, intraperitoneal, intratesticular, and intravenous routes. The tumors included adenocarcinomas and squamous cell carcinomas from inhalation, injection-site sarcomas, fibrosarcomas and squamous cell carcinomas from subcutaneous and intratracheal administration, and mesotheliomas from intraperitoneal injection (Stokinger 1981a). EPA's Carcinogen Assessment estimates a unit risk of 1.15 $(mg/kg/d)^{-1}$, based on human occupational (inhalation) exposure (CAG 1984).

The lowest observed effect level for nickel was reported by Schroeder and Mitchener (1971 as reported in NRC 1980). Rats administered 5 ppm nickel in drinking water in a three-generation study showed signs of toxicity including increased perinatal deaths and number of runts; the size of the litter decreased with each generation and few males were born in the third generation. These findings, however, have not been confirmed by other investigators (NRC 1980). Applying a 1000-fold safety factor to the lowest observed effect level in the Schroeder and Mitchener study would result in a toxicity reference dose of 0.5 μg/kg/d.

Whanger (1973, as cited in Stokinger 1981a) reported that a dose-related reduction in body weight gain occurred when rats were fed at 100, 500, and 1000 ppm nickel acetate in the diet. The application of a 1000-fold safety factor to the 100 ppm lowest observed effect level (in rats) would result in a toxicity reference dose of 6 μg/kg/d.

Nielson (1977 as reported in NRC 1980), indicates that generally 250 μg or more of nickel per gram of diet is required to produce signs of nickel toxicity in rats, mice, chicks, rabbits, and monkeys. Applying a 1000-fold safety factor to this level in rats would result in a toxicity reference dose of 15 μg/kg/d. According to NRC (1980), the daily oral intake averages of nickel by humans range from 165 to 500 μg (2.35-7.14 μ/kg/d); these averages are clearly in excess of the toxicity reference dose which would be derived based on Schroeder and Mitchener (1971) and Whanger 1973.

Based on the available toxicity data and knowledge of actual average human oral intake of nickel which have not been associated with adverse effects, an ADI of 15 μg/kg/d is recommended.

ZINC

Zinc is an essential element and is widely used in a number of therapeutic preparations. Most human and experimental animal data indicate that relatively large doses of zinc can be tolerated for long periods. However, toxic effects occur following acute exposures (USEPA 1980p). The most commonly reported effect of high-level zinc exposure in humans is reversible copper deficiency (USEPA 1980p). Metal fume fever is associated with acute occupational exposure to zinc oxide fumes. No adequate occupational exposure data are available to assign a minimum effective dose for metal fume fever (NIOSH 1975). Zinc poisonings in humans also result in nausea, vomiting, fever, anemia, and lethargy; these symptoms are reversible upon cessation of exposure (USEPA 1980p).

Rats exposed to zinc sulfate in drinking water for 14 months showed histopathological changes of several endocrine organs including hypertrophy of the adrenal cortex and changes in the pancreatic islets and pituitary gland (Aughey et al. 1977, as reported in USEPA, 1980p). Drinker et al. (1927, as reported in Stokinger 1981a) observed glycosuria in dogs and fibrous degeneration of the pancreas in cats administered 175 to 1000 mg zinc oxide per day in the diet. Sutton and Nelson (1937, as reported in Stokinger 1981a) reported that greater than 0.05 percent zinc in the diet of rats reduced their capacity to reproduce, and that 1 percent inhibited growth and caused severe anemia.

The zinc-containing fungicides Zineb [zinc ethylenebis (dithiocarbamate)] and Ziram [zinc dimethyl dithiocarbamate] were tested for teratogenic, reproductive, and mutagenic effects. At 50 mg/kg/d, the fungicides retarded the onset of pregnancy, and produced infertility, fetal resorption, and developmental abnormalities in rats. Zineb and Ziram were not mutagenic in a short-term assay with Drosophila (Ryazanova 1967, as reported in Stokinger 1981a). These positive results may not be relevant to zinc or other zinc compounds. No data were found on the carcinogenic potential of zinc.

Because zinc is an essential nutrient for humans, a recommended dietary allowance of 15 mg/d in adults has been established (NAS 1980 as reported in NRC 1980). This corresponds to 214 μg/kg/d assuming a 70 kg adult. An ADI of 2.1×10^{-1} mg/kg/d is therefore recommended for the oral and inhalation routes.

A-4 TOXICITY PROFILES FOR ACID GASES

HYDROGEN CHLORIDE

A wide variety of effects has been reported to be associated with exposure to hydrogen chloride, including necrosis of the tracheal and bronchial epithelia, pulmonary edema, atelectasis, emphysema, damage to the pulmonary blood vessels, liver lesions, nausea, and vomiting (Stokinger 1981b; NRC 1976). Henderson and Haggard (1943 as cited in ACGIH 1981) reported that exposure of men to 50-100 ppm HCl for one hour was barely tolerable, and 35 ppm caused irritation of the throat after a "short" exposure; 10 ppm was the maximum concentration tolerated for prolonged exposure. In workers exposed occupationally to concentrations of less than 1 ppm HCl, no statistically significant responses were seen (Patil et al. 1970 as cited in NRC 1976).

ASEL

ACGIH has set a ceiling limit of 5 ppm; this concentration should not be exceeded even instantaneously in occupational settings (ACGIH 1981). To estimate an acute NOEL for hydrogen chloride a safety factor of 10, to account for sensitive segments of a population, was applied to the concentration of 35 ppm (corresponding to LOEL in an exposed populations) and a safety factor of 2 was applied to convert the LOEL to a NOEL. This resulted in an acute safe exposure level (ASEL) of 1.75 ppm or 2.6 mg/m^3.

STPL

The National Academy of Sciences (1971) reviewed HCl and established guides for short-term exposure of the public. They conclude that the chief effect of inhalation exposure to HCl will be primary irritation of the moist mucous membranes of the upper respiratory tract. Prevention of the disagreeable and irritating odor of HCl was thus used as the basis for establishing short-term public exposure limits (STPL's). They assumed that greater than 5-10 ppm is disagreeable and irritating and should be avoided. The lowest concentration that could be smelled was between 1 and 10 ppm. The following STPL's were established by NAS and will be used for this report.

Short-Term Public Limits (STPL's)

10 min	6 mg/m^3 (4 ppm)
30 min	3 mg/m^3 (2 ppm)
60 min	3 mg/m^3 (2 ppm)
1 h daily	1 mg/m^3 (0.7 ppm)

The NAS (1971) emphasized that these levels are time-weighted averages not considered to present any health hazard. It should be recognized that excursions above these levels are likely to produce objectionable odors and/or irritation.

HYDROGEN CYANIDE

A considerable amount of information has been reported regarding human exposure to hydrogen cyanide. Four basic categories of effects result from acute exposure to cyanide: hematological, cardiovascular, neurological and metabolic (USEPA 1981a). The LC_{50} for a 30 second exposure ranges from 450 mg/m^3 in the mouse to 2500 mg/m^3 in the rat (McNamara, 1976 as cited in USEPA 1981a). Symptoms of exposure in workers exposed occupationally to cyanide include headache, weakness, change in taste and smell, irritation of throat, vomiting, abdominal colic, pericardial pain and nervous instability (ACGIH 1981).

Patty (1963 as cited in ACGIH 1981) compiled the following table regarding human response to acute hydrogen cyanide exposure:

Response	Concentrations mg/m^3
Immediate fatal	300
Fatal after 10 min	200
Fatal after 30 min	150
Fatal after ½ to 1 h or later or dangerous to life	120-150
Tolerated for ½ to 1 h without immediate or late effects	50-60
Slight symptoms after several hours	20-40

ASEL

The acute NOEL is estimated by applying safety factors of 10 for sensitive segments of the population, and 2 for conversion of a LOEL to a NOEL based on the occurrence of "slight symptoms" upon exposure to a concentration of 20 mg/m^3 for several hours. This calculation yields a recommended (ENVIRON) acute safe exposure level (ASEL) of 1.0 mg/m^3.

HYDROGEN FLUORIDE

Exposure to high concentrations of hydrogen flouride results in a variety of symptoms. When guinea pigs and rabbits were exposed to concentrations of approximately 50 mg/m^3 symptoms observed included signs of mild irritation, such as coughing and sneezing. At higher concentrations, signs of more severe irritation were observed, such as closing of the eyes, paroxysms of coughing and sneezing, slowing of respiration and discharge from nose and eyes (Machle et al, 1934 as cited in Stokinger 1981b). Rats were more sensitive to HF toxicity than rabbits, guinea pigs and dogs (Rosenholz et al, 1963 as cited in Stokinger 1981b). The LC_{50} for the rat is 4060 mg/m^3 for a 5 minute exposure, 1670 mg/m^3 for a 30 minute exposure and 1070 mg/m^3 for a 60 minute exposure. Signs of toxicity apart from irritation included pathological lesions in the liver and kidney (Rosenholz et al, 1963, as cited in Stokinger 1981b).

There is less information regarding acute exposure of humans to hydrogen flouride. The highest concentration tolerated for one minute was reported to be 98 mg/m^3. Symptoms at this concentration included conjunctival and respiratory irritation and smarting of the skin. At 26 mg/m^3 there was a mild smarting of the nose and eyes (Patty 1949 as cited in ACGIH 1981). Slightly longer exposure of humans, 6 h/d for 10 to 50 days, resulted in some irritation of nose and eyes at concentrations above 3 ppm. (Largent 1961 cited in ACGIH 1981), and ACGIH has set a TLV of 2.5 mg/m^3 (equivalent to about 3 ppm) hydrogen flouride (as F) for occupational exposure during a normal working week (ACGIH 1981). Applying a safety factor of 10 to account for sensitive segments of the population and, a safety factor of 2 to convert the acute LOEL of 26 mg/m^3 to a NOEL, based on mild irritation to the nose and eyes, results in an acute safe exposure level (ASEL) of 1.3 mg/m^3.

HYDROGEN SULFIDE

Acute exposure to hydrogen sulfide results predominantly in respiratory irritation. At extremely high concentrations (500-1000 ppm) unconsciousness and death may occur through respiratory paralysis (ACGIH 1981). Other symptoms of hydrogen sulfide exposure include CNS symptoms such as nervousness, cough, nausea, headache and insomnia as well as eye irritation (ACGIH 1981). The LC_{50} in mice is 673 ppm for a one hour exposure (NIOSH 1983).

The National Research Council (NRC 1979) compiled a table of human responses to various acute concentrations of hydrogen sulfide:

Effect	Concentration (mg/m^3)
Immediate collapse with respiratory paralysis	1400-2800
Strong nervous system stimulation, apnea	700-1400
Pulmonary edema	420-700
Olfactory paralysis	210-350
Threshold of serious eye injury	70-140
Offensive odor	4.2-7
Approximate threshold for odor	0.14-0.28

ACGIH has set a TLV for occupational exposure to hydrogen sulfide during a normal work week of 14 mg/m^3 (ACGIH 1981) based on the occurrence of headache, sleep disturbances, nausea, weight loss. Based on the threshold for eye injury of 70 mg/m^3 and applying a safety factor of 10 to protect sensitive segments of the population and 10 for the conversion of a LOEL to a NOEL for a serious effect, result in an estimated acute safe exposure level (ASEL) of 0.7 mg/m^3.

NITROGEN DIOXIDE

Exposure to nitrogen dioxide results in respiratory effects such as decreased lung capacity and bronchoconstriction. Other reported symptoms include a decreased resistance to disease and polycythemia (WHO 1977). The human olfactory threshold is 240 mg/m^3 while functional changes in the lungs of healthy human subjects begin after 10 minutes exposure to concentrations of 1300 mg/m^3 (WHO 1977). Increased reaction to a bronchoconstrictor was seen in asthmatic subjects after exposure to NO_2 concentrations of 190 mg/m^3 for one hour (Orehek et al. 1976 as cited in WHO 1977). Concentrations of 20 to 40 mg/m^3 have been reported to irritate the eyes, nose, and the respiratory tract (Patty 1949 as cited in ACGIH 1983).

Adley (1946 cited in ACGIH 1981) reported that "short" exposures to NO_2 averaging 25-38 ppm resulted in no observable effects and Norwood et al. (1966 cited in ACGIH) reported that 90 ppm for 30 min resulted in pulmonary edema. The USEPA (1982) summarized data showing that acute human exposures to nitrogen dioxide concentrations of approximately 300 ppm result in death; acute exposure to 150-200 ppm results in severe respiratory distress; acute exposures to 50-100 ppm result in reversible bronchiolitis; and exposures to 25-75 ppm result in reversible bronchial injury. Increased airway resistance was observed in healthy adults with a single 2-h NO_2 exposure of from 2.5-7.0 ppm, and altered airway resistance has been reported among healthy subjects exposed to concentrations somewhere in the range from 0.7-2.0 ppm (Suzuki and I Shikawa 1965 cited in USEPA 1982).

If two ppm of NO_2 (equivalent to about 4 mg/m^3) is considered a LOEL, a safety factor of 2 can be used to convert a LOEL of 4 mg/m^3 from the above study to a NOEL. Applying a safety factor of 10, to account for sensitive components of the population results in an acute safe exposure level (ASEL) of 0.2 mg/m^3.

SULFUR DIOXIDE

Sulfur dioxide is a strong irritant which will primarily affect the mucous membranes of the respiratory tract and the conjuctiva of the eye. Sulfur dioxide is very soluble in water and forms sulfuric acid in solution. The mucous present in nasal cavities can absorb large amounts of SO_2, therefore, the response to sulfur dioxide is more severe when inhaled through the mouth rather than the nose. The high water solubility also causes the upper respiratory tract to respond immediately to sulfur dioxide's local irritant properties through bronchoconstriction. Therefore, people with pre-existing respiratory problems, such as asthmatics and the elderly, are at greater risk of adverse effects of sulfur dioxide exposure (ACGIH 1981; Stoker and Seager 1976). Other symptoms associated with acute exposure to sulfur dioxide include excess salivation, moderate dyspnea, lacrimation, conjunctivitis, abdominal distension and lethargy (EPRI 1976).

Exposure of human subjects to 1 ppm of sulfur dioxide for 10 min caused a slight increase in lung air-flow resistance in only one of 11 subjects. Raising the concentration to 5 ppm caused a 72% increase (Frank et al., 1962). This response was not dose dependent but concentration dependent because increasing exposure period to 30 min did not cause an increase in the effect. A study by Tomono 1961 (as reported by ACGIH 1981) reported 1.6 ppm exposure was the lowest dose which produced bronchoconstriction in healthy subjects.

Stoker and Seager (1976) have summarized the effects of short-term SO_2 exposure on humans:

Effect	Concentration ppm
Immediate coughing	20
Eye irritation	10
Immediate throat irritation	8-12
Threshold for inducing reversible bronchial constriction in unimpaired individuals	1.6
Threshold for odor recognition	0.5
Threshold for taste recognition	0.3

ASEL

EPRI (1976) noted that in healthy males exposed to sulfur dioxide, 1 ppm is considered the threshold for bronchoconstrictor effects. A clear trend in increased pulmonary resistance is not seen until exposure concentrations reach several ppm. ACGIH has set a Threshold Limit Value of 2 ppm for occupational exposure to sulfur dioxide during a normal work week (ACGIH 1981). Using 1 ppm as the threshold for effects due to sulfur dioxide and applying a safety factor of 10 to protect sensitive segments of the population, the recommended acute safe exposure level (ASEL) is 0.1 ppm (0.26 mg/m^3).

STPL

NAS (1984) reviewed the data available on sulfur dioxide exposure and emphasized that changes in airway resistance are shortlived (about 5 min) at exposure levels of 30 ppm or less in (Attaway and Tyler 1976; Lawther et al.

1975 both as cited in NAS 1984). Although recovery of normal bronchial functioning is rapid in normal resting subjects, it may take 30-60 minutes after exercise, for asthmatics (Bates and Hazucha, 1973; Koenig et al., 1981; Gokemeijer et al. 1973; Lawther et al. 1975; all as cited in EPA 1982). The NAS (1984) estimated that 10-20% of the population will respond with bronchial hyperreactivity. OSHA (1983) recommended a TWA limit of 2 ppm. ACGIH (1980, 1983) recommended a TLV-TWA of 2 ppm for an 8-h period and a STEL of 5 ppm for 15 min. The reduction of the TLV from 5 to 2 ppm was based on available data and, in the opinion of the ACGIH committee, effects reported below 2 ppm were not serious enough to justify a lower limit.

NAS (1984) established emergency exposure limits (EELs) and continuous exposure limits (CELs) for sulfur dioxide. Emergency exposure limits relate to unpredictable single exposures, usually lasting 60 minutes or less, but never more than 24 hours, and which occur rarely in a person's lifetime. They reflect the acceptance of the occurrence of a nonincapacitating decrement in performance during emergencies. These EELs are established for defined occupational groups such as military personnel or space personnel, rather than the general public. Based on the reversibility of bronchoconstriction seen at levels below 30 ppm the NAS (1984) established the following EELs in 1984:

10-min EEL	30 ppm
30-min EEL	20 ppm
60-min EEL	10 ppm
24-h EEL	5 ppm

Because the population at risk in an occupational setting is generally younger and healthier than the normal population, short-term public exposure limits (STPLs) can differ somewhat from EELs. We therefore propose that the EELs established by NAS (1984) should be divided by a ten-fold safety factor to establish an STPLs. This would provide an adequate margin of safety for asthmatics and other respiratory compromised individuals. Our STPLs, for sulfur dioxide are listed below:

10-min STPL	3 ppm
30-min STPL	2 ppm
60-min STPL	1 ppm
24-hr STPL	0.5 ppm

REFERENCES

Altman, P.L., and Dittmer, D.S., eds. 1972. Biology Data Book, Second Edition. Federation of American Societies for Experimental Biology, Bethesda, Md.

Aviado, D.M. 1977. Preclinical pharamacology of toxicology and halogenated solvents and propellants. National Institute Drug Abuse Res. Monogr. Ser. 15:165-184.

American Conference of Governmental Industrial Hygienists (ACGIH). 1981. Documentation of the Threshold Limit Values. Fourth Edition. ACGIH, Cincinnati, Ohio

Bowman, R. Heironimus, M., and Barsotti, D. 1981. Locomotor hyperactivity in PCB-exposed Rhesus monkeys. Neurotoxicity 2:251-258

Buser, H.R. 1985. Formation, occurrence, and analysis of polyclorinated dibenzofurans, dioxins and related compounds. Env. Health Persp. 60:259-267.

Carcinogen Assessment Group (CAG). 1984. Relative Carcinogenic Potencies Among 53 Chemicals Evaluated by the Carcinogen Assessment Group as Suspect Human Carcinogens. From Draft EPA Health Assessment Document for Carbon Tetrachloride. September 1984

Carlsson, A. and Lindquist, T. 1977. as cited in: Health Assessment Document for Toluene. (Part 2). USEPA, 1982. EPA-600/8-82-008.

Clayton, G.D. and Clayton, F.E. 1981. Patty's Industrial Hygiene and Toxicology. Volume 2A. (John Wiley and Sons).

Clayton, G.D. and Clayton, F.E. 1981. Patty's Industrial Hygiene and Toxicology. Volume 2B. (John Wiley and Sons)

Clayton, G.D. and Clayton, F.E. 1982. Patty's Industrial Hygiene and Toxicology. Volume 2C. (John Wiley and Sons).

Cornish, H.H. 1980. Solvents and Vapors. In Toxicology, The Basic Science of Poisons, Second Edition (J. Doull, C. Klaasen and M. Amdur, eds.), Macmillan Publishing Co., New York

Deichmann, W.B. 1981. Halogenated cyclic hydrocarbons. In Patty's Industrial Hygiene and Toxicology, Third Edition (Clayton, G.D. and F.E. Clayton, eds.) Volume 2B, John Wiley and Sons, New York

Deichmann, W.B. and Keplinger, M.L. 1981. Phenols and phenolic compounds. In Patty's Industrial Hygiene and Toxicology, Third Edition (Clayton, G.D. and F.E. Clayton eds.) Volume 2A, John Wiley and Sons, New York

Doull, J., Klaasen, C.D., and Amdur, M.O. 1980. Casarett and Doull's Toxicology. Second Edition. MacMillan Publishing Co., Inc., New York

Electric Power Research Institute (EPRI). 1976. Sulfur Dioxides: Current Status of Knowledge EPRI EA-316. Palo Alto, California

Encyclopedia of Occupational Safety and Health (EOSH). 1971. Volume II: L-Z. International Labour Office. Geneva

Federal Register. 1983. Vol. 48 No. 10 p. 1884, January 14, 1983

Goldstein, J.A., Friesen, M., Linder, R.E., Hickman, P., Hass, J.R., and Bergman, H. 1977. Effects of pentachlorophenol on hepatic drug-metabolizing enzymes and porphyria related to contamination with chlorinated dibenzo-p-dioxins and dibenzofurans. Biochem. Pharm. 26:1549-1557.

Hammond, P.B., and Beliles, R.P. 1980. Metals. In: Doull, J., Klassen, C.D., and Amdus, M.O. eds. Toxicology: The Basic Science of Poisons. Macmillan Publishing Co., New York. pp. 409-467

Heller, V.G., and Purcell, L. 1938. Phenol-contaminated waters and their physiological action. J. Pharmacol. Exp. There. 63:99-107

Hoeltge, Inc. No date. Animal Care Equipment Catalogue. Cincinnati, Ohio.

International Agency for Research on Cancer (IARC). 1973. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol. 2: Some Inorganic and Organometallic Compounds. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1974a. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol. 4: Some Aromatic Amines, Hydrazine and Related Substances, N-Nitroso Compounds and Miscellaneous Alkylating Agents. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1974b. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol. 7: Some Anti-Thyroid and Related Substances, Nitrofurans and Industrial Chemicals. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1976. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Vol. 11: Cadmium, Nickel, Some Epoxides, Miscellaneous Industrial Chemicals and General Considerations on Volatile Anaesthetics. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1977. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Volume 15: Some Fumigants, the Herbicides 2,4-D and 2,4,5-T, Chlorinated Dibenzo-dioxins and Miscellaneous Industrial Chemicals. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1979a. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 19: Some Monomers, Plastics and Synthetic Elastomers and Acrolein. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1979b. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 20: Some Halogenated Hydrocarbons. World Health Organizations, Lyon, France

International Agency for Research on Cancer (IARC). 1980. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 23: Some Metals and Metallic Compounds. World Health Organization, Lyon, France

International Programmer on Chemical Safety (IPCS). 1984. Environmental. Health Criteria 32. Methylene Chloride. World Health Organization. Lyon, France

Kashimoto, T., Miyata, H., Fukushima, S., Kunita, N., Ohi, G. and Tung, T.C. 1985. PCBs, PCQs, and PCDFs in blood of Yusho and Yu-Cheng patients. Env. Health Persp. 59:73-78.

Kimbrough, R.D., Squire, R.A., Linder, R.E., Strandberg, J.D., Montali, R.J., and Burse, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. JNCI 55:1453-1456

Krasavage, W.J., O'Donoghue, J.L. and DiVincenzo, G.D. 1982. Ketones. In Patty's Industrial Hygiene and Toxicology, Third Edition (G.D. Clayton and F.E. Clayton, eds.) Volume 2C, John Wiley and Sons, New York

Lehman, A.J. 1959. Appraisal of the Safety of Chemicals in Foods, Drugs and Cosmetics. Association of Food and Drug Officials of the U.S.

Luster, M.I., Faith, R.E. and Lawson, L.D. 1979. Effects of 2,3,7,8-Tetrachlorodibenzofuran (TCDF) on the immune system in guinea pigs. Drug and Chem. Tox. 2(1 & 2):49-60.

Masuda, Y., Kuroki, H., Haraguchi, K., and Naagayama, J. 1985. PCB and PCDF congeners in the blood and tissues of Yusho and Yu-Cheng patients. Env. Health Persp. 59:53-58.

McNulty, W.P. 1985. Toxicity and fetotoxicity of TCDD, TCDF, and PCB isomers in rhesus macaques (Macaca mulatta). Env. Health Persp. 60:77-88.

Miyata, H., Fukushima, S., Kashimoto, T., and Kunita, N. 1985. PCBs, PCQs, and PCDFs in tissues of Yusho and Yu-Cheng patients. Env. Health Persp. 59:67-72.

Moore, H.R. 1979. Comparative toxicity of three halogenated dibenzofurans in guinea pigs, mice, and rhesus monkeys, In: Annals of The New York Academy of Science: Health Effects of Halogenated Aromatic Hydrocarbons. vol. 320. Nicholson and Moore, Eds. N.Y. Academy of Sciences. New York. Pp. 151-163.

National Academy of Sciences 1971. Guides for Short Term Exposures of the Public to Air Pollutants II. Guide for Hydrogen Chloride. Committee on Toxicology. National Technical Information Service APTD-0764

National Academy of Sciences (NAS). 1972. Lead: Airborne Lead in Perspective. Committee on the Biologic Effects of Atmospheric Pollutants, NAS, Washington, D.C.

National Academy of Sciences (NAS). 1977. Copper. Committee on Medical and Biologic Effects of Environmental Pollutants, Division of Medical Sciences, Assembly of Life Sciences, National Research Council, Washington, D.C.

National Academy of Sciences 1984, Emergency and Continuous Exposure Limits for Selected Airborne Contaminants Vol. 2. Committee on Toxicology. National Academy Press. Washington, D.C.

National Institute for Occupational Safety and Health (NIOSH). 1975. Criteria for a Recommended Standard: Occupational Exposure to Zinc Oxide. DHEW Publication No. 76-104

National Institute for Occupational Safety and Health (NIOSH). 1976. Criteria for a Recommended Standard -- Occupational Exposure to Methylene Chloride. PB81-117017.

National Institute for Occupational Safety and Health (NIOSH). 1976. Criteria for a Recommended Standard: Occupational Exposure to Isopropyl Alcohol. DHEW Publication No. 76-142

National Institute for Occupational Safety and Health (NIOSH). 1977. Occupational Disease: A Guide to Their Recognition. U.S. Department of H.E.W., P.H.S., C.D.C., NIOSH. DHEW (NIOSH) Publication No. 77-181

National Institute for Occupational Safety and Health (NIOSH). Criteria for a recommended standard... Occupational exposure to refined petroleum solvents. DHEW (NIOSH) Publication No. 77-192. 1977

National Institute for Occupational Safety and Health (NIOSH). 1980. Registry of Toxic Effects of Chemical Substances. 1979 Edition. Volume 1. DHHS (NIOSH) Publication No. 80-111.

National Institute for Occupational Safety and Health (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances (RTECS). 1981-82 Ed. Volumes 1-3. DHHS (NIOSH) Publication No. 83-107

National Research Council (NRC) 1976. Medical and Biological Effects of Environmental Pollutants. Chlorine and Hydrogen Chloride. National Academy of Sciences, Washington, D.C.

National Research Council (NRC). 1977. Board on Toxicology and Environmental Health Hazards. Safe Drinking Water Committee. Drinking Water and Health. Volume 1. National Academy Press, Washington, D.C.

National Research Council (NRC) 1979. Medical and Biological Effects of Environmental Pollutants. Hydrogen Sulfide. University Park Press. Baltimore

National Research Council 1979a. Criteria for Short Term Exposures to Air Pollutants. National Academy of Sciences Committee on Toxicology. Washington, D.C.

National Research Council (NRC). 1980. Board on Toxicology and Environmental Health Hazards. Safe Drinking Water Committee. Drinking Water and Health. Volume 3. National Academy Press, Washington, D.C.

National Research Council (NRC). 1981. Formaldehyde and Other Aldehydes. Committee on Aldehydes, Board of Toxicology and Environmental Health Hazards, Assembly of Life Sciences

National Research Council (NRC). 1983. Board on Toxicology and Environmental Health Hazards. Safe Drinking Water Committee. Drinking Water and Health. Volume 5. National Academy Press, Washington, D.C.

National Research Council (NRC). 1983. Drinking Water and Health. Volume 5. National Academy of Sciences, Washington, D.C.

National Toxicology Program (NTP). 1984a. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Benzene (CAS No. 71-43-2) in F344/N Rats and B6C3F1 Mice (Gavage Studies). Draft Report. NTP-TR-289. NIH Publication No. 84-2545. NTP-84-072

National Toxicology Program (NTP). 1984b. Management Status Report. October 24, 1984

Poland, A., Knutson, J., and Glover, E. 1983. A consideration of the mechanism of action of 2,3,7,8-TCDD and related halogenated aromatic hydrocarbons. In: Human and Environmental Risk of Chlorinated Dioxins and Related Compounds. Tucker, Young, and Gray, eds. Plenum Press, New York.

Ontario Ministry of the Environment (OMOE). 1985. Polychlorinated dibenzo-p-dioxin [TCDDs] and polychlorinated dibenzo furans [PCDFs]. Scientific Criteria Document for Standard Development No. 4-84. Prepared for Intergovernmental Relations and Hazardous Contaminants Branch.

Ontario Ministry of the Environment (OMOE). 1984. Preliminary Hearing Document on Proposals for the Regulation of Mobile PCB Destruction Facilities in Ontario. Prepared for presentation to the Commission of Inquiry on the Regulatory Control of Mobile PCB Destruction Facilities. Pp. 37-39.

Reggiani, G. and Bruppacher, R. 1985. Symptoms signs and findings in humans exposed to PCBs and their derivatives. Env. Health Persp. 60:225-232.

Reinhardt, C.F. and Brittelli, M.R. 1981. Heterocyclic and Miscellaneous Nitrogen Compounds. In Patty's Industrial Hygiene and Toxicology, Third Edition, (G.D. Clayton and F.E. Clayton eds.) Volume 2A, John Wiley and Sons, New York

Rowe, V.K. and McCollister, S.B. 1982. Alcohols. In Patty's Industrial Hygiene and Toxicology, Third Edition, (G.D. Clayton and F.E. Clayton, eds.) Volume 2C, John Wiley and Sons, New York

Sandmeyer, E.E. 1981. Aromatic Hydrocarbons. In Patty's Industrial Hygiene and Toxicology, Third Edition (G.D. Clayton and F.E. Clayton, eds.) Volume 2B, John Wiley and Sons, New York

Stoker, H.S. and Seager, S.L. 1976. Environmental Chemistry: Air and Water Pollution. Second Edition. Scott Foresman and Company. Glenview, Illinois

Stokinger, H.E. 1981a. The Metals. In Patty's Industrial Hygiene and Toxicology, Third Revised Edition, (Clayton, G.D. and Clayton F.E. eds.) Volume 2A, John Wiley and Sons, New York

Stokinger, H.E. 1981b. The Halogens and the Nonmetals Boron and Silicon. In Patty's Industrial Hygiene and Toxicology, Third Edition, (Clayton, G.D. and Clayton, F.E., eds.) Volume 2B, John Wiley and Sons, New York

Tiesinger, J. 1948. J. Ind. Hyg. Tox. 30:58

Torkelson, T.R. and Rowe, V.K. 1981. Halogenated aliphatic hydrocarbons. In Patty's Industrial Hygiene and Toxicology, Third Edition, (Clayton, G.D. and F.E. Clayton, eds.) volume 2B, John Wiley and Sons, New York

Tseng, W.P. 1977. Effects and Dose-response Relationships of Skin Cancer and Blackfoot Disease with Arsentic. Environ. Health Perspect. 19:109-119.

Tseng, W.P., Chu, H.M., How, S.W., Fong, J.M., Lin, C.S., and Yeh, S. 1968. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463.

U.S. Environmental Protection Agency (USEPA). 1977. Air Quality Criteria for Lead. Office of Research and Development, USEPA, Washington, D.C. PB-280-411

U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria Documents, Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. (e.g., Ambient Water Quality Criteria for Phthalate Esters, EPA/440/5-80-067. PB81-117780)

U.S. Environmental Protection Agency (USEPA). 1980a. Ambient Water Quality Criteria for Acrylonitrile. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-017. PB81-117285

U.S. Environmental Protection Agency (USEPA). 1980b. Ambient Water Quality Criteria for Benzene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-018. PB81-117293

U.S. Environmental Protection Agency (USEPA). 1980c. Ambient Water Quality Criteria for Chloroform. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-057. PB 81-117681

U.S. Environmental Protection Agency (USEPA). 1980d. Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. Draft report. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1980e. Ambient Water Quality Criteria for Polychlorinated Biphenyls. Office of Water Regulations and Standards, Criteria and Standards, Division, Washington, D.C. EPA 440/5-80-068. PB81-117798.

U.S. Environmental Protection Agency (USEPA). 1980f. Ambient Water Quality Criteria for Phenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-066. PB81-117772

U.S. Environmental Protection Agency (USEPA). 1980g. (revised 1983). An Exposure and Risk Assessment for Tetrachloroethylene. Final Draft Report

U.S. Environmental Protection Agency (USEPA). 1980h. Ambient Water Quality Criteria for Toluene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-75. PB81-117855

U.S. Environmental Protection Agency (USEPA). 1980i. Ambient Water Quality Criteria for Arsenic. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-021. PB81-117327

U.S. Environmental Protection Agency (USEPA). 1980j. Ambient Water Quality Criteria for Cadmium. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-025. PB81-117368

U.S. Environmental Protection Agency (USEPA). 1980k. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-035. PB81-117467

U.S. Environmental Protection Agency (USEPA). 1980l. Ambient Water Quality Criteria for Copper. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-036. PB81-117475

U.S. Environmental Protection Agency (USEPA). 1980m. Ambient Water Quality Criteria for Lead. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-057. PB81-117681

U.S. Environmental Protection Agency (USEPA). 1980n. Ambient Water Quality Criteria for Mercury. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-058

U.S. Environmental Protection Agency (USEPA). 1980o. Ambient Water Quality Criteria for Nickel. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-080. PB81-117715

U.S. Environmental Protection Agency (USEPA). 1980p. Ambient Water Quality Criteria for Zinc. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-079. PB81-117897

U.S. Environmental Protection Agency (USEPA). 1981. Advisory Opinion for Dichloromethane (Methylene Chloride), Office of Drinking Water, Draft.

U.S. Environmental Protection Agency (USEPA). 1981a. An Exposure and Risk Assessment for Cyanide. Office of Water Regulations and Standards, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1981b. An Exposure and Risk Assessment for Trichloroethylene. Final Draft Report, March 1981

U.S. Environmental Protection Agency (USEPA). 1981c. An Exposure and Risk Assessment for Nickel. Final Draft Report. U.S. EPA Contract 68-01-5949, Task 3. Office of Water Regulations and Standards (WH-553), Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1981d. An Exposure and Risk Assessment for Arsenic (Final Draft Report). Office of Water Regulations and Standards, Monitoring and Data Support Divisions, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1982. Health Effects Assessment Summary for 300 Hazardous Organic Constituents in Support of Regulatory Impact Analysis of the Land Disposal Branch (LDB) and Interim Final Incinerator Regulations of the Technical Branch (TB) of the Office of Solid Wastes, OSWER/EPA, Draft, September 20, 1982

U.S. Environmental Protection Agency (USEPA). 1983. Health Assessment Document for Acrylonitrile. Final Report. EPA-600/8-22-007F.

U.S. Environmental Protection Agency (USEPA). 1983. Health Assessment Document for Dichloromethane (Methylene Chloride). Office of Health and Environmental Assessment, Washington, D.C. December 1983, External Review Draft, EPA-600/8-82-004B.

U.S. Protection Agency. 1984. Research and Development, Summary of Current Acceptable Daily Intakes (ADIs) for Oral Exposure. Environmental Criteria and Assessment Office, Cincinnati, Ohio.

U.S. Environmental Protection Agency (USEPA). 1984. Summary of Current Acceptable Daily Intakes (ADIs) of Oral Exposure. Internal Review Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. February 1984

U.S. Environmental Protection Agency (USEPA). 1984. Air Quality Criteria for Lead. Review draft. Environmental Criteria and Assessment office. EPA-600/8-83-028B

U.S. Environmental Protection Agency (USEPA). 1984. Health Assessment Document for Chromium. Final Report. Publication No. EPA-600-8/83-014F. August, 1984

U.S. Environmental Protection Agency (USEPA). 1984. Health Assessment Document for Beryllium. Review Draft. EPA-600/8-84-026A

U.S. Environmental Protection Agency (USEPA). 1985. Health Assessment Document for Chloroform. Review Draft. EPA-600/8-84-004F

U.S. Environmental Protection Agency (USEPA). 1985. Health Assessment Document for Dichloromethane (Methylene Chloride)

Van Den Berg, M., Olie, K., and Hutzinger, O. 1985. Polychlorinated Dibenzofurans (PCDFs). Tox. Env. Chem. 9:171-217.

World Health Organization (WHO). 1977. Environmental Health Criteria. Oxides of Nitrogen. World Health Organization. Geneva

World Health Organization (WHO). 1983. Styrene, IPCS International Program on Chemical Safety. Environmental Health Criteria 26. World Health Organization, Geneva. p.32.

APPENDIX B

SELECTION OF SURROGATE CHEMICALS

CONTENTS

	Page
B.1 INTRODUCTION	B-1
B.2 BASIS FOR HAZARD INDEX	B-6
Release Potential	B-8
Toxicity	B-10
Quantity	B-14
B.3 RESULTS OF SURROGATE SELECTION	B-15
Selection of Surrogate Chemicals	B-15
Calculation of Mass Fractions	B-22
B.4 LIMITATIONS OF SURROGATE SELECTION PROCEDURE	B-24
REFERENCES	B-28

LIST OF TABLES

Page

B-1 OWMC Waste Classes B-2

B-2 Waste Categories Included in the Organic Constituent Selection Procedure B-3

B-3 Concentrations of Organic Contaminants in Waste Oils B-5

B-4 Basis for Hazard Indices B-7

B-5 Vapor Pressure Scores B-9

B-6 Acute Toxicity Scores B-11

B-7 Chronic Toxicity Scores B-13

B-8 Quantity Scores B-15

B-9 Environmental Parameters and Associated Scores B-16

B-10 Basis for Chronic Toxicity Scores B-17

B-11 Chemicals Scoring the Highest Hazard Index B-20

B-12 Mass Fractions for Fugitive and Incinerator Emissions B-23

B-13 Mass Fractions for Truck Spills and Tank Farm Fires B-25

B-14 Summary of Usable Data Points from Canviro Data Base B-27

B.1 INTRODUCTION

The surrogate selection procedure was conducted to identify hazardous waste constituents to be included in the modeling of human health risk. To compensate for some of the limitations in the waste data base, surrogate chemicals were selected to represent conservatively the most hazardous wastes potentially to be disposed of at the OWMC facility. This appendix describes in detail the procedure used to select the surrogate chemicals and estimate their concentrations in the organic waste streams to be managed by the facility. The chemicals used to model risks associated with emissions of metals and products of the incineration process, e.g. acid gases and products of incomplete combustion (PICs), such as dioxins and furans, were selected from empirical data compiled by the Ontario Research Foundation (ORF, 1986) and are not addressed here (see Appendix C for empirical data for metals, acid gases and dioxins/furans).

The starting point for the selection of surrogate organic constituents was the waste characterization data base developed by Canviro Associates (Canviro, 1985). The data base was developed from a survey of the types and quantities of special wastes generated by a sample of Ontario manufacturers. Waste in this survey has been categorized using the University of California at Davis (UCD) classification scheme, whereby waste streams are grouped in UCD categories according to similarities in chemical constituents and physical state (see OWMC, 1982). The UCD categories are organized further into broader waste classes, which are listed in Table B-1. The surrogate selection process was used to identify the most hazardous constituent present in the waste streams of each UCD category of concern. In effect, each waste stream was represented by the most hazardous constituent, and each UCD category was represented by the most hazardous waste stream in that category. In this way, a surrogate chemical was selected to represent each UCD category in the modeling of environmental releases.

TABLE B-1

OWMC WASTE CLASSES

Class	Description
1	Organic sludges and still bottoms
2	Solvents and organic solutions
3	Oils and greases
4	Oil and water mixtures
5	Organic and oily residuals
6	Heavy metal solutions and residuals
7	Miscellaneous chemicals and products
8	Paint and organic residuals
9	Aqueous solutions and organics
10	Anion complexes
11	Sludges and inorganic residuals
12	Pesticide and herbicide waste
13	PCB wastes
14	Clean up residuals

Because this surrogate selection procedure was designed to identify organic constituents, the procedure was applied only to the UCD categories which were considered to contain significant concentrations of organic chemicals. The UCD classes and categories included in the surrogate selection procedure (i.e., those waste streams considered to contain significant quantities of organic constituents) are shown in Table B-2. In the case of Class 4, Oil and Water Mixtures, adequate data were not available from the Canviro data base to permit quantitative characterization of the surrogate chemicals. An alternative data base (Franklin Associates, 1983), which surveyed organic contaminants in waste oils, was used therefore to characterize this waste class. This data base was developed for the United States Environmental Protection Agency and was based on a relatively large number of samples of waste lubricants. Quantitative data were presented for concentrations in waste oils of a range of commonly used solvents, PCBs and

TABLE B-2

WASTE CATEGORIES INCLUDED IN THE ORGANIC CONSTITUENT SURROGATE SELECTION PROCEDURE

OWMC Class	UCD Category	Description
1	247	Halogenated still bottoms
	248	Still bottoms
	252	Solid or sludge tannery waste
	257	Phenolic wastes
	261	Polymeric resins
	263	Other polymeric wastes
	446	Degreasing sludge
	523	Tank bottom sediments
2	211	Halogenated solvents with heavy metals
	212	Halogenated solvents with other metals
	213	Other halogenated solvents
	214	Solvents without metal/non-metals
	216	Other non-halogenated solvents
	217	Unspecified solvents
	221	Organic liquid metals and halogens
	222	Organic liquids with halogens only
	223	Organic liquids with heavy metals
	224	Other organic liquids

TABLE B-2 (cont'd)

WASTE CATEGORIES INCLUDED IN THE ORGANIC CONSTITUENT SURROGATE SELECTION PROCEDURE

OWMC Class	UCD Category	Description
3	281	Waste oil and mixed oil
4	283	Oily mud, sediment and water
5	264	Thermoset wastes
	282	Oily tank bottoms
	284	Oily acidic solids
	285	Oily alkaline solids
	286	Oil/water separator sludge
	288	Oily wastes with heavy metals
	289	Oily wastes with water
	447	Tetraethyl lead sludge
7	145	Contaminated brine
	152	Inorganic solid chemicals
	243	Pharmaceutical wastes
	245	Off spec. or aged organics
	416	Ink sludge
	513	Lab. waste chemicals
8	254	Adhesives or glue
	258	Waste tar and residues
	262	Latex wastes
	454	Paint sludge
	455	Paint sludge with no heavy metals
9	225	Aqueous solutions, <10% organic residues
	226	Aqueous solutions, <10% halogenated residues
	227	Aqueous solutions, <10% organic residues
	228	Aqueous solutions, <10% halogenated residues
12	241	Pesticides and wastes
	532	Contaminated equipment and containers
13	242	PCB oil and high strength liquids
14	512	Spill cleanup
	521	Spent cartridge filters
	528	Pathological wastes
	531	Contaminated rags

other chemicals, and are summarized in Table B-3. While it was recognized that these data may not fully represent the organic contaminants to be found in Ontario wastes, in the absence of other sources of information they were considered a useful approximation.

TABLE B-3

CONCENTRATIONS OF ORGANIC CONTAMINANTS IN WASTE OILS

Contaminant	Total Samples Analyzed for Contaminant	Total Samples with Detectable Concentrations	Average of Values (mg/L)
PCBs	113	20	127
Benzo[a]pyrene	1	0	N/A
Naphthalene	1	1	440
1,1,1-Trichloroethane	95	7	1025
Trichloroethylene	101	90	527
Tetrachloroethylene	86	79	448
Dichlorodifluoromethane	65	50	380
Benzene	1	1	160
Toluene	2	2	1260

Source: Franklin Associates (1983)

The waste characterization data described above were used in a series of separate ranking schemes developed for each source of environmental release modeled, i.e., fugitive emissions, truck spills, incinerator emissions, and tank farm fires. Each ranking scheme generated a hazard index to represent the degree of hazard associated with each chemical constituent of the waste streams of concern. The constituent in each UCD category with the highest hazard index was selected as the surrogate for that UCD category. In general, the hazard rankings were based on three factors: (1) the toxicity of the constituent; (2) the potential for the constituent to be released to the environment; and (3) the quantity of chemical available for release. The hazard index for each constituent was calculated by multiplying a set of scores for the following three factors:

$$HI = R \times T \times Q$$

where HI = hazard index

R = release potential

T = toxicity

Q = quantity of chemical available for release

The hazard index resulting from the multiplication of these scores was thus designed to provide a systematic means for comparing the relative hazard presented by waste constituents.

B.2 BASIS FOR HAZARD INDEX

To represent each of the general factors comprising the hazard indices described above, chemical specific parameters were chosen. The specific parameters representing toxicity and release potential were different, depending upon the source of release under consideration. The basis for the hazard indices derived for each source of environmental release and the specific parameters used to derive the hazard indices are summarized in Table B-4. Operationally, a scoring scheme was developed for each parameter used in the calculation of the hazard indices. The scores for the appropriate parameters representing toxicity, release potential and quantity were then multiplied resulting in the hazard index. The following sections describe the basis for the parameters and scoring systems used to calculate the hazard indices.

TABLE B-4

BASIS FOR HAZARD INDICES

Source of Release	Hazard Index Basis	Parameter
Fugitive Emissions	Release Potential	Vapor Pressure
	Toxicity	Chronic Toxicity
	Quantity	Chemical Quantity
Incinerator Emissions	Toxicity	Chronic Toxicity
	Quantity	Chemical Quantity
Truck Spill	Release Potential	Vapor Pressure
	Toxicity	Acute Toxicity
	Quantity	Chemical Quantity
Tank Farm Fire	Release Potential	Heat of Vaporization
	Toxicity	Acute Toxicity
	Quantity	Chemical Quantity

RELEASE POTENTIAL

Vapor Pressure

The parameter used to represent release potential for the truck spill and fugitive emission releases was selected by examining the equations used to calculate emission rates. For example, the equation used to calculate the emission rates for truck spills was (see Appendix D, Volume 2):

$$Q_i = \frac{(Kg_i)(A)(P_i)(M_i)(x_i)}{(R)(T)}$$

where Q = emission rate for species, g/s

Kg_i = the gas mass transfer coefficient, m/s

A = area of spill, m^2

P_i = vapor pressure of the chemical, Pa

M_i = molecular weight of chemical, g/mol

R = gas constant, $(Pa.m^3)/(mol.K)$

T = temperature, K

x_i = mole fraction of species i in waste

The chemical-specific parameters in the above equation are gas mass transfer coefficient, vapor pressure, molecular weight and mole fraction. Similarly, for the fugitive emissions, the emission rates are typically a function of vapor pressure, molecular weight and mole fraction. For example, the losses from storage tanks are given by (see Appendix C):

$$Q_i = (4.91 \times 10^{-6})(P_i)(M_i)(x_i)$$

where: Q = emission rate for species i, g/s

P_i = vapor pressure of chemical, Pa

M_i = molecular weight of chemical, g/mol

x_i = mole fraction of species i in waste

Mole fraction appears in both equations and is treated separately under the heading of "quantity". Of the other parameters, vapor pressure was considered to be the primary parameter differentiating between emission rates for various chemicals. Vapor pressure scores then were assigned on a scale of 1 to 10 as shown in Table B-5.

TABLE B-5

VAPOR PRESSURE SCORES

Vapor Pressure (mm Hg)	Score
>100	10
50-100	9
10-50	8
5-10	7
1-5	6
0.5-1	5
0.1-0.5	4
0.05-0.01	3
0.01-0.05	2
<.01	1

In the release of chemicals via incinerator emissions, all organic wastes were assumed to be in the vapor phase at the high temperatures involved in the incinerator process. For this reason, release potential was not included in the calculation of the hazard index for the incinerator emissions source of release.

Heat of Vaporization

Similar to the truck spill and fugitive emission releases, the parameter representing release potential for tank farm fire releases was selected by review of the emission rate equation (see Section 3.4, Volume 1):

$$Q_i = \frac{(21\ 000)\ (F)\ (Aw)^{0.82}}{\Delta H_i}$$

where Q = emission rate for species i, lb/h

F = insulation factor, 1.0 for a base vessel

Aw = wetted surface area of tank, m^2

ΔH_i = heat of vaporization for species i, BTU/lb

Since the heat of vaporization is the only chemical-specific parameter in this equation, the reciprocal of heat of vaporization ($1/\Delta H$) was used to represent release potential in the calculation of the Hazard Index.

TOXICITY

Acute Toxicity

Acute toxicity was chosen to represent toxicity for the truck spill and tank farm fire releases. In both of these scenarios, a large quantity of chemical is released over a relatively short period of time. Thus, acute effects of the chemicals would be of primary concern.

The acute toxicity score was based on LD_{50} values. The LD_{50} represents that dose which is, on average, lethal to fifty percent of the test population following a single exposure (Doull et al., 1980). For consistency, the lowest LD_{50} reported for a mammalian species was used in the ranking.

The LD_{50} values were assigned scores according to the classes defined by Gosselin, Smith and Hodge (1984) as shown in Table B-6.

TABLE B-6

ACUTE TOXICITY SCORES

Score	Description	LD_{50}(mg/kg)
1	Practically non toxic	>15000
2	Slightly toxic	500-15000
3	Moderately toxic	500-5000
4	Very toxic	50-500
5	Extremely toxic	5-50
6	Super toxic	<5

Chronic Toxicity

For releases via fugitive and incinerator emissions, chronic toxicity was chosen to represent toxicity. In both of these scenarios, relatively small emissions of chemicals occur over a long period of time. Thus, chronic effects of the chemicals would be of primary concern.

The chemicals were ranked according to their chronic toxicity using Acceptable Daily Intakes (ADIs) for noncarcinogens and Unit Cancer Risks (UCRs) for carcinogens as derived in Appendix A. Because these measures of risk are expressed using different parameters, it was necessary to develop a method for scoring both carcinogenic and noncarcinogenic compounds using the same scale. This was accomplished by converting the UCRs to a Risk Specific Dose (RSD) using the following approach.

By assuming that the dose-response curve for carcinogens is a straight line at very low doses, an RSD can be calculated for carcinogens using the formula:

$$RSD = \frac{\text{Level of Risk}}{\text{UCR}}$$

To derive an RSD from a UCR, a set of assumptions can then be made regarding the level of risk considered to be acceptable, and the level of risk associated with noncarcinogenic ADIs. Using these assumptions, a level of risk can be chosen which will enable the direct comparison of carcinogenic and noncarcinogenic toxic potential.

Carcinogens, under the assumptions used to derive UCRs, present a non-zero risk at all exposure levels above zero. Nevertheless, it is commonly accepted in regulatory decision-making that for all carcinogens there is an exposure range under which the magnitude of the risk is very small. In many cases, lifetime risks falling below the range of 10^{-5} to 10^{-6} have been considered very small. These probabilities reflect highly unlikely events, especially when it is further considered that they are predicted using extrapolation methods designed to produce upper bound estimates.

ADIs are thought to reflect conditions of exposure to noncarcinogens that are without risk. It is not possible, however, to claim that an ADI carries absolutely no risk for the human population. At best, all that can be claimed for an ADI is that any associated risk is likely to be very small. No means are available to quantify this "very small" risk. In the absence of evidence to the contrary and to provide a systematic means for comparing carcinogenic and noncarcinogenic compounds (so as to avoid having to create two different toxicity scales), it was assumed that the "risk" associated with an ADI is in the same range (10^{-5} to 10^{-6}) as that considered to be "very small" for carcinogens. This assumption should not be used outside the context of this specific scoring objective. Neither should it be taken to mean that the actual risk to a target population at an ADI exposure level is in the range assumed here. This assumption has been used only to provide a systematic means of aligning and comparing the risks associated with carcinogenic and noncarcinogenic compounds for this study.

It is further assumed that exposure at an RSD for a noncarcinogen will ensure that there is no more than a 10^{-6} (1 in 1 000 000) risk of a toxic effect occurring. An RSD derived for carcinogens using a risk level of 10^{-6} will represent the dose that will give rise to the same maximum lifetime risk (10^{-6}). Carcinogens and noncarcinogens can then be directly compared because they will have been adjusted for potency and represent the same risk level. Using this methodology, scores were assigned according to order of magnitude changes in the RSDs or ADIs as shown in Table B-7.

TABLE B-7

CHRONIC TOXICITY SCORES

RSD or ADI (mg/kg/day)	Score
$\leq 10^{-7}$	9
$>10^{-7}$ - $\leq 10^{-6}$	8
$>10^{-6}$ - $\leq 10^{-5}$	7
$>10^{-5}$ - $\leq 10^{-4}$	6
$>10^{-4}$ - $\leq 10^{-3}$	5
$>10^{-3}$ - $\leq 10^{-2}$	4
$>10^{-2}$ - $\leq 10^{-1}$	3
$>10^{-1}$ - ≤ 1	2
>1	1

QUANTITY

For all sources of release, the quantity of the constituent was used to represent quantity. The quantity of each constituent was derived from the waste stream data in the Canviro database with the exception of Class 4 wastes which were characterized using the data from Franklin Associates. The objective of this procedure was to estimate annual volumes of specific constituents of waste streams associated with each UCD category. Aggregation of constituent quantities was restricted to wastes within UCD categories because only within these categories were wastes considered generically similar. Thus, the following three-step procedure was applied to each of the UCD categories listed in Table B-2.

Step (1) Calculate quantity of each individual waste stream. Because of a concern that quantities of individual waste streams projected in the waste profile might underestimate the actual quantities, the approach adopted was to give equal status to each waste stream. Individual waste stream quantity was thus approximated as the extrapolated quantity of the pertinent UCD category in the waste profile divided by the number of wastes identified in the data base for that category.

Step (2) Calculate constituent quantity for each waste stream. Constituent quantity was calculated by multiplying the constituent concentration reported in the Canviro data base by the waste stream quantity estimated in Step 1.

Step (3) Generate aggregated constituent quantity for each UCD category. Quantities of each constituent were summed for all waste streams within each UCD category generating an aggregated quantity of each hazardous constituent present in the UCD category.

The aggregated quantities obtained were then placed on a scale of 1-6 as shown in Table B-8. In this manner, a numerical quantity score was obtained for those chemicals appearing as waste constituents in the Canviro data base.

TABLE B-8

QUANTITY SCORES

Quantity (t/a)	Score
>500	6
101-500	5
51-100	4
11-50	3
1-10	2
<1	1

B.3 RESULTS OF SURROGATE SELECTION

SELECTION OF SURROGATE CHEMICALS

The results of the surrogate selection process are summarized in a series of tables:

(1) The parameters and associated scores used to select surrogate chemicals are shown in Table B-9.

(2) The basis for the chronic toxicity values (ADIs or RSDs) are summarized in Table B-10.

(3) The chemicals scoring the highest hazard index within each UCD class are shown in Table B-11. We note that in the case of the chronic emissions associated with fugitive releases and incineration, all UCD categories were assumed to contribute to the overall emissions. The surrogate for each UCD category, therefore, was selected for use in the subsequent modeling of emissions.

TABLE B-9

ENVIRONMENTAL PARAMETERS AND ASSOCIATED SCORES*

Compound	RSD or ADI (mg/kg/day)	Score	LD50 (mg/day)	Score	Vapor Pressure (mm Hg)	V.P. Temp. (C)	Score	Heat of Vaporization (kJ/Mole)	Score
Acetone	2.86E+00	1	9750 (7)	2	2.40E+02 (1)	25	10	31.3 (1)	0.032
Acrylonitrile	4.17E-06	7	27 (7)	5	1.03E+02 (1)	25	10	32.6 (1)	0.031
Benzene	1.92E-05	6	3800 (7)	3	9.39E+01 (1)	25	9	30.7 (1)	0.033
Chlorobenzene	1.40E-02	3	2830 (7)	3	1.19E+01 (1)	25	8	36.5 (1)	0.027
Chloroform	1.33E-05	6	800 (7)	3	1.96E+02 (1)	25	10	29.7 (1)	0.034
Cresols	1.13E-01	2	242 (7)	4	1.43E-01 (1)	25	4	46.7 (1)	0.021
o-Dichlorobenzene	9.00E-02	3	414 (7)	4	1.39E+00 (1)	25	6	39.7 (1)	0.025
Dichlorodifluoromethane	8.00E-01	2	47500 (7)	1	4.87E+03 (3)	25	10	24.4 (1)	0.041
Ethanol	4.95E-02	3	5560 (7)	2	5.88E+01 (1)	25	9	38.7 (1)	0.026
Ethylene glycol	1.00E+00	1	7500 (7)	2	5.02E-02 (2)	20	3	52.5 (1)	0.019
Fluorocarbon 113	5.02E+01	1	43000 (7)	1	3.32E+02 (1)	25	10	27.5 (1)	0.036
Formaldehyde	8.93E-05	6	260 (7)	4	3.83E+03 (1)	25	10	23 (1)	0.043
Glycerol	1.70E-02	3	7750 (7)	2	1.37E-05 (1)	25	1	61.1 (1)	0.016
Hexachlorobenzene	5.99E-07	8	10000 (7)	2	1.10E-05 (3)	20	1	64 (6)	0.016
n-Hexane	5.20E+00	1	28710 (7)	1	1.51E+02 (1)	25	10	28.8 (1)	0.035
Hydrazine	2.56E-07	8	91 (7)	4	1.42E+01 (1)	25	8	44.8 (1)	0.022
Isopropyl alcohol	6.40E-02	3	5840 (7)	2	4.52E+01 (1)	25	8	39.8 (1)	0.025
Kerosene	2.40E-02	3	20000 (7)	1	5.00E+00 (5)	25	7	24 (6)	0.042
Methyl ethyl ketone	2.00E-02	3	2740 (7)	3	9.41E+01 (1)	25	9	31.2 (1)	0.032
Methyl isobutyl ketone	1.04E-01	2	2080 (7)	3	1.96E+01 (1)	25	8	35.5 (1)	0.028
Methylene chloride	1.59E-03	4	167 (7)	4	4.36E+02 (1)	25	10	27.9 (1)	0.036
Napthalene	2.57E-01	2	1780 (7)	3	8.70E-02 (3)	25	3	43.3 (1)	0.023
PCB	2.30E-07	8	1010 (7)	3	2.42E-04 (4)	25	1	54 (6)	0.019
Petroleum distillates	1.12E-01	2	1240 (7)	3	4.00E+00 (5)	25	6	31 (6)	0.032
Phenol	1.10E-01	2	300 (7)	4	5.00E-01 (2)	25	5	45.6 (1)	0.022
Pyridine	9.00E-03	4	891 (7)	3	2.03E+01 (1)	25	8	35.1 (1)	0.028
Styrene	9.50E-01	2	316 (7)	4	5.89E+00 (1)	25	7	36.8 (1)	0.027
Tetrachloroethylene	2.86E-05	6	8100 (7)	2	1.89E+01 (1)	25	8	34.7 (1)	0.029
Tetraethyl lead	2.80E-07	8	15 (7)	5	1.50E-01 (2)	20	4	54 (8)	0.019
Toluene	4.2[illegible]E-01	2	5000 (7)	3	2.74E+01 (1)	25	8	33.1 (1)	0.030
1,1,1-Trichloroethane	6.25E-04	5	750 (7)	3	2.40E+01 (1)	25	8	33.3 (1)	0.030
Trichloroethylene	5.26E-05	6	4920 (7)	3	7.45E+01 (1)	25	9	31.3 (1)	0.032
Xylene	2.29E+00	1	4300 (7)	3	7.79E+00 (1)	25	7	36.4 (1)	0.027

---

* References are in parentheses:

(1) Reid et al., 1977
(2) Verschueren, 1983
(3) Mabey et al., 1982
(4) NAS, 1979
(5) GE Manual, 1980
(6) Calculated
(7) NIOSH, 1983
(8) Weast, 1984
(9) CRC, 1984

TABLE B-10

BASIS FOR CHRONIC TOXICITY SCORES

COMPOUND	RSD or ADI (mg/kg/day)	SCORE	BASIS	REFERENCE
Acetone	2.86	1	USEPA ADI based on TLV associated with minor irritation.	USEPA, 1984
Acrylonitrile	4.17×10^{-6}	7	USEPA unit risk value based on human occupational exposure.	CAG, 1984
Benzene	1.92×10^{-5}	6	USEPA unit risk based on human occupational exposure.	CAG, 1984
Chlorobenzene	1.40×10^{-2}	3	USEPA ADI based on rat subchronic NOEL; higher doses caused histopathological changes in the liver.	USEPA, 1984
Chloroform	1.33×10^{-5}	6	USEPA unit risk value based on oral animal study	CAG, 1984
Cresols	0.113	2	USEPA ADI based on TLV	USEPA, 1984
O-Dichlorobenzene	9.0×10^{-2}	3	USEPA ADI based on rat subchronic oral NOEL higher doses caused increased liver and kidney weights and hepatic necrosis	USEPA, 1984
Ethanol	4.95×10^{-2}	3	Rat 90 day inhalation NOEL; higher doses induced cirrhois of the liver	See Appendix A
Ethylene glycol	1.0	1	Rat 2 year oral NOEL; higher doses produced calcification of the kidneys and renal injury	Morris et al as cited in Clayton and Clayton 1981-
Fluorocarbon 113	50.2	1	Rabbit, 12-24 month NOEL; higher doses produced fatty infiltration of the liver	Reinke 1962 as cited in ACGIH 1981

TABLE B-10 (cont'd)

BASIS FOR CHRONIC TOXICITY SCORES

COMPOUND	RSD OR ADI (mg/kg/day)	SCORE	BASIS	Reference
Formaldehyde	8.93×10^{-5}	6	USEPA unit risk based on nasal carcinomas	See Appendix
Glycerol	1.7×10^{-2}	3	TLV	ACGIH 1981
Hexachlorobenzene	5.99×10^{-7}	8	USEPA unit risk based on oral animal study	CAG, 1984
n-Hexane	5.2	1	Mouse, 1 yr NOEL; higher doses produced neurotoxic effects	Miyagaki as cited in Spencer et al. 1980
Hydrazine	2.56×10^{-7}	8	USEPA VCR value based on occurence of adenocarcinomas o the lung	USEPA, 1982
Isopropyl alcohol	6.4×10^{-2}	3	Human 6 week oral NOEL; higher doses in a different study caused CNS effects such as ataxia prostration and narcosis	See Appendix A
Kerosene	2.4×10^{-2}	3	NIOSH recommended standard	NIOSH 1977 as cited in Clayton and Clayton 1981-1982
Methyl ethyl ketone	2.00×10^{-2}	3	USEPA ADI based on subchronic rat inhalation NOEL; higher doses caused teratogenic effects in animals	USEPA, 1984
Methyl isobutyl ketone	0.104	2	USEPA ADI based on TLV associated with liver and kidney damage in rats	USEPA, 1984
Methylene chloride	1.59×10^{-3}	4	USEPA unit risk based on oral animal study	CAG, 1984
Naphthalene	0.257	2	USEPA ADI based on TLV; higher doses caused headache, eye injury and kidney damage	USEPA, 1984

TABLE B-10 (cont'd)

BASIS FOR CHRONIC TOXICITY SCORES

COMPOUND	RSD OR ADI (mg/kg/day)	SCORE	BASIS	Reference
PCB-1254	2.3×10^{-7}	8	USEPA unit risk based on animal study	CAG, 1984
Phenol	0.11	2	USEPA ADI based on rat subchronic to chronic oral LOEL associated with slight kidney damage	USEPA, 1984
Petroleum Distillates	1.12×10^{-1}	2	Monkey, 90 day NOEL associated with a decline in body weight	See Appendix A
Pyridine	9.0×10^{-3}	4	Rat 6 month inhalation NOEL associated with increased liver weight	See Appendix A
Styrene	0.95	2	Rat 185 day oral NOEL; higher doses caused liver and kidney weight deviations	See Appendix A
Tetrachloroe-ethylene	2.86×10^{-5}	6	USEPA unit risk value based on oral animal study	CAG, 1984
Tetraethyl lead	2.8×10^{-7}	8	Rat 20 week oral LOEL associated with histological changes in liver, pancreas, renal, endocrine and nervous system	See Appendix A
Toluene	0.428	2	USEPA ADI value based on rat chronic oral NOEL; higher doses have not been tested	USEPA, 1984
Trichloroethylene	5.26×10^{-5}	6	USEPA unit risk based on oral animal study	CAG, 1984
1,1,1-Trichloro-ethane	6.25×10^{-4}	5	USEPA unit risk based on oral animal study	CAG, 1984
Xylene	2.29	1	USEPA ADI based on TLV; higher inhaled doses caused maternal toxicity in a rat teratology study	USEPA, 1984

TABLE B-11

CHEMICALS SCORING THE HIGHEST HAZARD INDEX

UCD Category	Fugitive Emissions	Score	Incinerator Emissions	Score	Truck Spills	Score	Tank Farm Fire	Score
211	Trichloroethylene	270	Tetrachloroethylene Trichloroethylene	30 30	Methylene chloride	200	Methylene chloride	0.717
212	Trichloroethylene	162	Trichloroethylene Tetrachloroethylene	18 18	Trichloroethylene	81	Trichloroethylene	0.288
213	Chloroform	240	1,1,1-Trichloroethane	25	Methylene chloride	200	Methylene chloride	0.717
214	Petroleum distillates	72	Petroleum distillates	12	Petroleum distillates	108	Petroleum distillates	0.581
215	Toluene	80	Toluene	10	Toluene	120	Toluene	0.453
216	Benzene	108	Benzene	12	Toluene	96	Petroleum distillates	0.484
217	Ethanol	135	Ethanol	15	Ethanol	90	Ethanol	0.258
224	Acrylonitrile	140	Hydrazine	16	Acrylonitrile	100	Acrylonitrile	0.307
225	Formaldehyde	120	Formaldehyde	12	Formaldehyde	80	Formaldehyde	0.348
227	Methyl ethyl ketone	54	Toluene Methyl ethyl ketone	6 6	Toluene	72	Toluene	0.272
241	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290
242	PCB	32	PCB	32	PCB	12	PCB	0.222
243	Ethanol	81	Ethanol	9	Ethanol Petroleum distillates	54 54	Petroleum distillates	0.290
245	Formaldehyde	120	Ethanol Formaldehyde	12 12	Formaldehyde	80	Formaldehyde	0.348
247	Methylene chloride	80	Methylene chloride	8	Methylene chloride	80	Methylene chloride	0.287
248	Ethanol	135	Ethanol	15	Petroleum distillates	108	Petroleum distillates	0.581
254	1,1,1-Trichloroethane Methylene chloride	120 120	1,1,1-Trichloroethane	15	Methylene chloride	120	Methylene chloride	0.430
257	Formaldehyde	120	Phenol Formaldehyde	12 12	Phenol	120	Phenol	0.526
261	Styrene	56	Styrene Cresols	8 8	Styrene	112	Styrene	0.435
262	Formaldehyde	60	Formaldehyde	6	Formaldehyde	40	Formaldehyde	0.174
263	Acrylonitrile	140	Acrylonitrile	14	Acrylonitrile	100	Acrylonitrile	0.307
264	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290

TABLE B-11 (cont'd)

CHEMICALS SCORING THE HIGHEST HAZARD INDEX

UCD Category	Fugitive Emissions	Score	Incinerator Emissions	Score	Truck Spills	Score	Tank Farm Fire	Score
281	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290
283	Trichloroethylene Benzene	108 108	PCB	16	Trichloroethylene Benzene	54 54	Benzene	0.195
286	Phenol	20	Phenol	4	Phenol	40	Phenol	0.175
289	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290
416	Ethanol	81	Ethanol	9	Ethanol	54	Petroleum distillates	0.194
446	Trichloroethylene	270	Tetrachloroethylene Trichloroethylene	30 30	Trichloroethylene	135	Trichloroethylene	0.479
447	Tetraethyl lead	32	Tetraethyl lead	8	Tetraethyl lead	20	Tetraethyl lead	0.093
454	Petroleum distillates	72	Petroleum distillates	12	Petroleum distillates	108	Petroleum distillates	0.581
455	Petroleum distillates	48	Petroleum distillates	8	Petroleum distillates	72	Petroleum distillates	0.387
513	Pyridine	96	Pyridine	12	Pyridine	72	Petroleum distillates	0.290
521	Toluene	16	Toluene	2	Toluene	24	Petroleum distillates	0.097
523	Formaldehyde	120	Formaldehyde	12	Formaldehyde	80	Formaldehyde	0.348
531	Petroleum distillates	60	Petroleum distillates	10	Petroleum distillates	90	Petroleum distillates	0.484

In the case of episodic releases associated with truck spills and the tank farm fire, the situation is different, since any given event will only involve a single UCD category, i.e., that category being transported or stored. Consequently, for the episodic releases, only the constituent with the highest hazard index (methylene chloride as shown in Table B-11) was selected for subsequent modeling.

CALCULATION OF MASS FRACTIONS

The final step in the surrogate selection process was the generation of mass fractions to be used in the calculation of air emissions (see Appendix C for emission equations). Again, different mass fractions were generated for each source of environmental release. For the chronic emissions, i.e., incinerator and fugitive emissions, it was assumed for the purposes of modeling that all of the waste streams are processed together essentially as a mixture. The mass fraction, therefore, was calculated by summing all of the estimated quantities for each surrogate chemical and dividing this sum by the total quantity of waste processed by the facility. In the case of a "tie" (two or more chemicals producing the same hazard index), the quantity of each surrogate was adjusted accordingly. For example, if two chemicals tied for a given UCD category, the quantity associated with the UCD category was split equally between the two chemicals. The mass fractions for fugitive and incinerator emissions are shown in Table B-12.

For the truck spill scenario, it was assumed that each waste stream would be transported separately. Therefore, the mass fraction was calculated simply as the average concentration of the surrogate chemical in that UCD category. Similarly, for the tank farm fire, it was assumed that each waste stream could

TABLE B-12

MASS FRACTIONS FOR FUGITIVE AND INCINERATOR EMISSIONS

Fugitive Emissions		Incinerator Emissions	
Compound	Mass Fraction	Compound	Mass Fraction
Acrylonitrile	1.01E-03	Acrylonitrile	3.37E-04
Benzene	2.03E-03	Benzene	2.02E-03
Chloroform	8.42E-03	Cresols	1.51E-02
Ethanol	5.88E-02	Ethanol	5.56E-02
Formaldehyde	1.04E-03	Formaldehyde	1.30E-03
Methyl ethyl ketone	3.37E-04	Hydrazine	3.37E-04
Methylene chloride	3.47E-03	Methyl ethyl ketone	1.68E-04
PCB	1.06E-03	Methylene chloride	1.01E-04
Petroleum distillates	6.04E-02	PCB	1.08E-03
Phenol	5.05E-05	Petroleum distillates	6.04E-02
Pyridine	2.83E-03	Phenol	4.93E-03
Styrene	2.83E-02	Pyridine	2.83E-03
Tetraethyl lead	1.68E-06	Styrene	1.41E-02
Toluene	8.00E-03	Tetrachloroethylene	1.03E-02
1,1,1-Trichloroethane	3.37E-03	Tetraethyl lead	1.68E-06
Trichloroethylene	1.98E-02	Toluene	9.67E-03
		1,1,1-Trichloroethane	1.82E-02
		Trichloroethylene	9.90E-03

be the only waste stream present in a holding tank. Thus, the mass fraction was calculated as the average concentration of the surrogate chemical in each UCD category. The mass fractions for the tank farm fire and truck spills are shown by UCD category in Table B-13.

B.4 LIMITATIONS OF SURROGATE SELECTION PROCEDURE

In applying the ranking scheme, the following limitations of the data were noted:

(1) The data base was developed from a partial survey of Ontario industries and may not be fully representative of the types of hazardous wastes generated in Ontario. In some cases, the sample of waste addressed within a UCD category by Canviro was only a small fraction of the total waste projection for that UCD category, making any direct extrapolation of quantities or chemical types questionable. For example, UCD category 247 includes four waste streams totalling 9.32 tonnes per annum compared with a total of 600 tonnes per annum projected for the facility.

(2) The amount of quantitative waste composition data suitable for use in a ranking scheme was limited. This limitation arises in two ways. In some cases, hazardous waste constituents were identified, but without quantitative concentrations. In other cases, where quantitative concentrations were provided, the constituents were described only in generic terms (e.g., aromatic hydrocarbons) as opposed to a specific chemical compound. Where feasible, this latter problem was addressed by making reasonable assumptions about

TABLE B-13

MASS FRACTIONS FOR TRUCK SPILLS AND TANK FARM FIRES

Truck Spill		
SCD Category	Compound	Mass Fraction
211	Methylene chloride	0.62
212	Trichloroethylene	0.49
213	Methylene chloride	0.68
214	Petroleum distillates	0.63
215	Toluene	0.79
216	Toluene	0.4
217	Ethanol	0.58
224	Acrylonitrile	0.1
225	Formaldehyde	0.01
227	Toluene	0.2
241	Petroleum distillates	0.8
242	PCB	0.79
243	Ethanol	0.18
243	Petroleum distillates	0.5
245	Formaldehyde	0.04
247	Methylene chloride	0.01
248	Petroleum distillates	0.55
254	Methylene chloride	0.4
257	Phenol	0.29
261	Styrene	0.28
262	Formaldehyde	0.02
263	Acrylonitrile	0.005
264	Petroleum distillates	0.56
281	Petroleum distillates	0.32
283	Trichloroethylene	0.000527
283	Benzene	0.00016
286	Phenol	0.005
289	Petroleum distillates	0.07
416	Ethanol	0.45
446	Trichloroethylene	0.28
447	Tetraethyl lead	0.01
454	Petroleum distillates	0.22
455	Petroleum distillates	0.18
513	Pyridine	0.42
521	Toluene	0.001
523	Formaldehyde	0.001
531	Petroleum distillates	0.33

Tank Farm Fire		
SCD Category	Compound	Mass Fraction
211	Methylene chloride	0.62
212	Trichloroethylene	0.49
213	Methylene chloride	0.68
214	Petroleum distillates	0.63
215	Toluene	0.79
216	Petroleum distillates	0.51
217	Ethanol	0.58
224	Acrylonitrile	0.1
225	Formaldehyde	0.01
227	Toluene	0.2
241	Petroleum distillates	0.8
242	PCB	0.79
243	Petroleum distillates	0.5
245	Formaldehyde	0.04
247	Methylene chloride	0.01
248	Petroleum distillates	0.55
254	Methylene chloride	0.4
257	Phenol	0.29
261	Styrene	0.28
262	Formaldehyde	0.02
263	Acrylonitrile	0.005
264	Petroleum distillates	0.56
281	Petroleum distillates	0.32
283	Benzene	0.00016
286	Phenol	0.005
289	Petroleum distillates	0.07
416	Petroleum distillates	0.1
446	Trichloroethylene	0.28
447	Tetraethyl lead	0.01
454	Petroleum distillates	0.22
455	Petroleum distillates	0.18
513	Petroleum distillates	0.99
521	Petroleum distillates	0.001
523	Formaldehyde	0.001
531	Petroleum distillates	0.33

the meaning of the generic terms, e.g., "phenols", were assumed to be phenol and "chlorinated ethylenes" were assumed to be a 50/50 mixture of trichloroethylene and tetrachloroethylene. While this was feasible in some instances, some categories of wastes could only be characterized in a very general way, e.g. herbicides and pesticides.

The net effect of this lack of specificity was to limit the number of waste streams that could be quantitatively characterized to approximately 60% of the total number (390) containing organic chemicals addressed by the Canviro survey. The amount of usable data contained in the Canviro data base is summarized by waste stream class in Table B-14.

The total number of data points (each corresponding to a constituent) in the Canviro data base is shown in the second column. The third column lists the number of those data points for which an associated concentration was cited. Finally, the number of data points that were ultimately used is listed in the fourth column. In cases where hazardous waste constituents were identified without quantitative concentrations, waste streams with concentrations in the same category were used to extrapolate to the deficient streams. In one case (class 13), the extrapolation procedure resulted in the number of usable data points exceeding the number of data points with available concentrations. As can be seen, 425 of 2659 (or about 16%) of the data points listed in the data base were usable for the purposes of this assessment.

TABLE B-14

SUMMARY OF USABLE DATA POINTS FROM THE CANVIRO DATA BASE

Waste Stream Class	Total Data Points	Data Points With Conc.	Usable Data Points
1	320	185	51
2	668	421	242
3	403	343	7
4	DATA TAKEN FROM FRANKLIN ASSOCIATES (1983)		
5	313	260	9
7	270	131	33
8	347	207	42
9	33	30	7
12	17	12	1
13	27	8	19
14	261	102	14
TOTAL	2659	1699	425

REFERENCES

American Conference of Government Industrial Hygienists (ACGIH). 1981. Documentation of the Threshold Limit Values. 4th ed. Supplemental Documentation, 1983. ACGIH, Cincinnati, Ohio.

Canviro Consultants, Ltd. 1985. Market Assessment Program: Survey of 1000 Industrial Establishments. Prepared for Ontario Waste Management Corporation.

Carcinogen Assessment Group (CAG). 1984. Relative Carcinogenic Potencies Among 53 Chemicals Evaluated by the Carcinogen Assessment Group as Suspect Carcinogens. From Draft EPA Health Assessment Document for Carbon Tetrachloride. September.

Clayton, G.D. and Clayton, F.E. 1981-1982. Patty's Industrial Hygiene and Toxicology. Third Revised Edition. Vols. 2A, 2B, and 2C: Toxicology. John Wiley and Sons, New York.

CRC. 1984. Handbook of Chemistry and Physics, 64th Edition. Ed. Robert C. Weast, Ph.D.

Doull et al. 1980. Casarett and Doull's Toxicology. Second Edition. MacMillan Publishing Co., Inc., New York.

Franklin Associates, Ltd. 1983. Revised Draft Report on Waste Oil Management and Composition. USEPA Office of Solid Waste.

General Electric Co. (GE). 1980. Material Safety Data Sheets. Nielsen, J.M. ed.

Gosselin, Smith, & Hodge. 1984. Clinical Toxicology of Commercial Products. Fifth Edition. Williams and Wilkins, Baltimore.

Mabey, W.R., Smith, J.H., Podoll, R.T., Johnson, H.L., Mill, T., Chou, T.W., Gates, J., Partridge, I.W., Jaber, H., and Vandenberg, O. 1982. Aquatic Fate Process Data for Organic Priority Pollutants. Prepared by SRI International for U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Monitoring and Data Support Division, Washington, D.C. EPA Contracts 68-01-3867 and 68-03-2981.

National Academy of Sciences (NAS). 1979. Polychlorinated Biphenyls. Environmental Studies Board Commission on Natural Resources, National Research Council. Washington, D.C.

National Institute for Occupational Safety and Health (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances (RTECS), 1981-1982 ed. Vols. 1-3. DHHS (NIOSH) Publication No. 33-107

Ontario Research Foundation. 1985. Emissions Related Studies: Preliminary Estimates of Air Emissions. In preparation for Ontario Waste Management Corporation.

Ontario Waste Management Corporation. 1982. Facilities Development Process. Phase 1 Report. September.

Reid, R.C., Prausnitz, J.M. and Sherwood, T.K. 1977. Properties of Gases and Liquids. McGraw-Hill Book Co., New York

Spencer, P.S., Schaumburg, H.S., Sabri, M.I. and Veronesi, B. 1980. The enlarging view of hexacarbon neurotoxicity. CRC Crit. Rev. in Tox. 7:279-357

Stiver, W. and Mackay, D. 1982. Spill Technology Newsletter, January/ February. As cited in Technical Information for Problem Spills. Draft Introduction Manual.

United States Environmental Protection Agency. 1982. Health Effects Assessment Summary for 300 Hazardous Organic Constituents. In support of Regulatory Impact Analysis of the Land Disposal Branch and Interim Final Incinerator Regulations of the Technical Branch of the Office of Solid Waste. Draft, September.

USEPA, 1984. Summary of Current Acceptable Daily Intakes (ADIs) of Oral Exposure. Environmental Criteria and Assessment Office, Cincinnati, Ohio.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. 2nd ed. Van Nostrand Reinhold Co., New York.

APPENDIX C

ESTIMATION OF ENVIRONMENTAL RELEASES

CONTENTS

C.1 INTRODUCTION
C.1.1 Emission Sources C-1
C.1.2 Assumptions for Calculations C-2

C.2 WASTE TRANSPORTATION C-8

C.3 BULK WASTE UNLOADING AND TRANSFER C-11
C.3.1 Organic Waste Reception - Normal Operation C-13
C.3.2 Organic Waste Reception - Failure C-15
C.3.3 Inorganic Waste Reception - Normal Operation C-15
C.3.4 Inorganic Waste Reception - Failure C-16
C.3.5 Routine Spills C-17

C.4 DRUM UNLOADING AND TRANSFER C-20
C.4.1 Drum Opening C-21
C.4.2 Drum Transfer C-23

C.5 WASTE STORAGE TANKS C-27
C.5.1 Organic Waste Storage C-28
C.5.2 Inorganic Waste Storage C-30
C.5.3 Tank Farm Fire C-31

C.6 PHYSICAL CHEMICAL TREATMENT C-32
C.6.1 Dissolved Air Flotation C-33
C.6.2 Emulsion Breaking C-35
C.6.3 Cyanide Treatment C-36
C.6.4 Sulfide Treatment C-38
C.6.5 Mixing of Incompatible Wastes C-40

C.7 INCINERATION C-40
C.7.1 Controlled Stack Emissions C-41
C.7.2 Incinerator Scrubber Failure C-45

C.8 SUMMARY OF RESULTS C-46

REFERENCES C-52

TABLES

		Page
C-1	Mole Fractions for Estimation of Emission Rates	C-5
C-2	Chemical Property Data for Estimation of Emission Rates	C-7
C-3	Stack Emission Estimates - Metals	C-43
C-4	Stack Emission Estimates - Acid Gas	C-44
C-5	Stack Emission Estimates - PICs	C-45
C-6	Stack Emission Estimates - Scrubber Failure	C-46
C-7	Fugitive Emission Rates at 150 000 t/a	C-47
C-8	Fugitive Emission Rates at 300 000 t/a	C-48
C-9	Incinerator Stack Emission Estimates	C-49
C-10	Emission Estimates for Upset Scenarios	C-50

C.1 INTRODUCTION

C.1.1 EMISSION SOURCES

The proposed OWMC facility will consist of four major components: an incinerator, a physical-chemical treatment plant, a solidification plant and an engineered landfill, along with the associated transportation, reception and storage facilities (See Monenco, 1985). Air emissions will be generated from these sources under four possible scenarios: normal operating conditions, facility upset conditions, on-site accidents and off-site transportation accidents. The focus of the emission estimates was primarily in gaseous pollutants (in particular volatile organic chemicals) released under these scenarios, although consideration was also given to heavy metals and organics associated with particulate emissions from the incinerator operation. Fugitive dust emissions were not considered a significant contribution to the risk assessment, and are not discussed further here.

The magnitude of the emissions of concern was estimated by a combination of empirical observations from comparable facilities (See ORF, 1985; Abkovitz et al., 1984) and by engineering calculations (See Monenco, 1985; Dillon, 1984; Dillon, 1985). The empirical data and the calculation methods are described here for the following facility emission sources:

(1) Transportation

- Accidental spill

(2) Waste Handling and Storage

- Waste unloading and transfer
- Routine spills
- Drum opening and transfer
- Waste storage
- Tank farm fire

(3) Physical/Chemical Treatment

- Dissolved air flotation
- Emulsion breaking
- Cyanide treatment
- Sulfide treatment
- Mixing of incompatible wastes

(4) Incineration

- Controlled stack emissions
- Scrubber failure

C.1.2 ASSUMPTIONS FOR CALCULATIONS

The calculations for the emissions provided by the engineering consultants (Monenco, 1985) were developed based on a single component system, i.e. an individual chemical. In order to extend these single component system estimates to the multiple component system found in a waste mixture, Raoult's Law for ideal vapor-liquid equilibria was used to approximate the behavior of hazardous wastes mixtures.

Raoult's Law for multi-component mixtures is based on the following assumptions:

- the gas phase behaves as an ideal gas
- the liquid phase behaves as an ideal liquid
- the fugacity of the mixture is insensitive to variations in pressure

Raoult's Law is given by:

$$y_i = \frac{x_i P_i}{P_o}$$

where Y_i = mole fraction of component i in the gas phase

x_i = mole fraction of component i in the liquid phase

P_i = vapor pressure of component i, mm Hg

P_o = ambient pressure, mm Hg

While it is recognized that there is currently a lack of either empirical or theoretical correlations capable of accurately predicting the vapor-liquid equilibrium for multi-component mixtures of complex hydrocarbons, considering the uncertainty with which the composition of the waste streams is known, the error introduced in assuming Raoult's Law is considered to be relatively small. The effective partial pressure of each component of the waste mixture to be handled at the OWMC facility was thus approximated from the mole fraction of each component of the mixture. The mole fractions for each component were derived from the mass fractions presented in Appendix B, Selection of Surrogate Chemicals.

Calculation of the mole fraction in the waste stream requires a knowledge of the molecular weights of the other waste constituents. Given the varying composition of waste streams, it is difficult to make any such determination precisely. Based on consideration of the range of constituents expected to be present in the waste streams to be received at the OWMC facility, an average molecular weight for the components of the mixture other than the constituent of concern was assumed to be 150.

The mole fraction was then estimated as:

$$X = \frac{MF_x(MW_{av})}{MW_x}$$

where X = mole fraction of constituent, dimensionless

MF_x = mass fraction of the constituent, dimensionless

MW_x = molecular weight of the constituent, g/mol

MW_{av} = average molecular weight for the waste stream, g/mol

The average molecular weight is defined as:

$$MW_{av} = (MF_x)(MW_x) + (1 - MF_x)(150)$$

so that the mole fraction can now be defined as:

$$X = \frac{MF_x\ [(MF_x)(MW_x) + (1 - MF_x)(150)]}{MW_x}$$

The mole fractions calculated in this way are presented in Table C-1.

TABLE C-1

MOLE FRACTIONS FOR ESTIMATION OF EMISSION RATES

Chemical Constituents	Fugitive Emission Mole Fraction	Incinerator Emission Mole Fraction
Trichloroethylene	2.25E-02	1.13E-02
Perchloroethylene	0.00E+00	9.33E-03
Chloroform	1.06E-02	0.00E+00
1,1,1-Trichloroethane	3.79E-03	2.04E-02
Hydrazine	0.00E+00	1.58E-03
Acrylonitrile	2.85E-03	9.52E-04
Methylene Chloride	6.12E-03	1.78E-04
Pyridine	5.36E-03	5.36E-03
Benzene	3.89E-03	3.88E-03
Toluene	1.30E-02	1.57E-02
Phenol	8.05E-05	7.84E-03
Formaldehyde	5.19E-03	6.49E-03
Petroleum Distillates	6.91E-02	6.91E-02
Tetraethyl Lead	7.79E-07	7.79E-07
PCB	4.85E-04	4.94E-04
Ethanol	1.84E-01	1.74E-01
Styrene	4.04E-02	2.02E-02
Methyl ethyl ketone	7.01E-04	3.49E-04
Cresols	0.00E+00	2.09E-02

Other data inputs used in the calculation of emissions are summarized in Table C-2. The vapor pressures were estimated at different temperatures using the Antoine equation (Lyman, 1982). The vapor pressures at 10 °C (annual average) were necessary for fugitive emission calculations from activities conducted outdoors, such as waste unloading, transfer and storage. Indoor activities such as drum opening and transfer were assumed to be conducted at 25 °C, while emulsion breaking is carried out in a heated unit at a temperature of 70 °C.

Another assumption used generically in the calculation of fugitive emissions was the efficiency of adsorption/scrubbing in the plant ventilation systems. Fugitive emissions from the facility will generally be recovered or captured by adsorption/scrubbing which removes the bulk of the contaminants. The removal efficiency depends on the properties of the contaminant, and, for the purposes of the risk assessment, was conservatively assumed to be 90%.

Finally, two scenarios were considered for the capacity and configuration of the facility: an initial throughput of 150 000 t/a of wastes operating with a single incinerator (of capacity 30 000 t/a); and the ultimate capacity of 300 000 t/a with two incinerators (with a combined capacity of 60 000 t/a). While it might at first be expected that the difference between the two scenarios would be a simple doubling of emissions in going from 150 000 t/a to 300 000 t/a, for the fugitive emissions this is not necessarily the case. Some of the sources contributing to fugitive emissions exhaust to the incinerator feed air, others vent to the atmosphere, either directly or via an adsorber. When the incinerator is out of operation for maintenance those sources that typically exhaust to the incinerator, vent to the atmosphere via

TABLE C-2

CHEMICAL PROPERTY DATA FOR ESTIMATION OF EMISSION RATES**

CHEMICAL CONSTITUENT	MOLEC WEIGHT (g/mol)	P_i* @ 10 C (mm-Hg)	P_i @ 25 C (mm-Hg)	P_i @ 70 C (mm-Hg)	DIFF IN AIR (cm^2/s)
Trichloroethylene	131.389	3.51E+01(2)	7.38E+01(2)	4.39E+02(2)	8.75E-02
Perchloroethylene	165.834	6.20E+00(1)	1.40E+01(4)	1.42E+02(2)	7.97E-02
Chloroform	119.378	9.82E+01(2)	1.93E+02(2)	9.81E+02(2)	8.88E-02
1,1,1-Trichloroethane	133.405	4.40E+01(1)	1.00E+02(5)	7.60E+02(3)	7.94E-02
Hydrazine	32.05	7.10E+00(1)	1.60E+01(5)	1.44E+02(2)	1.90E-01
Acrylonitrile	53.064	5.07E+01(2)	1.05E+02(2)	5.97E+02(2)	1.06E-01
Methylene chloride	84.933	2.29E+02(2)	4.33E+02(2)	1.99E+03(1)	1.04E-01
Pyridine	79.102	8.50E+00(2)	2.06E+01(2)	1.66E+02(2)	9.50E-02
Benzene	78.114	4.52E+01(2)	9.45E+01(2)	5.48E+02(2)	9.32E-02
Toluene	92.141	1.23E+01(2)	2.82E+01(2)	2.03E+02(2)	8.49E-02
Phenol	94.113	1.40E-01(1)	5.00E-01(5)	1.00E+01(3)	8.50E-02
Formaldehyde	30.026	2.35E+03(2)	3.83E+03(2)	1.25E+04(2)	1.70E-01
Petroleum distillates	130	1.78E+00(1)	4.00E+00(6)	N/A	7.57E-02
Tetraethyl lead	323.44	7.00E-02(1)	1.52E-01(5)	1.00E+01(3)	1.00E-02
PCB	328.4	3.43E-05(1)	7.71E-05(4)	1.37E-01(2)	4.00E-02
Ethanol	46.069	2.33E+01(2)	5.86E+01(2)	5.38E+02(2)	1.18E-01
Styrene	104.16	4.40E+00(1)	6.12E+01(2)	6.04E+00(2)	7.50E-02
Methyl ethyl ketone	72.1	4.52E+01(2)	1.00E+02(3)	5.53E+02(2)	8.94E-02
Cresols	108.13	3.23E-02(1)	1.75E-01(5)	5.00E+00(3)	8.10E-02

* P_i symbolizes Vapor Pressure

** References are in parentheses

(1) Calculated

(2) Reid and Prausnitz, 1977

(3) CRC, 1984

(4) Mabey et al., 1982

(5) Verscheuren, 1983

(6) GE Manual, 1980

an adsorber. If however, there are two incinerator units, the source is essentially eliminated, since one incinerator unit is expected to be operational at all times. The only exception to this would occur if during the scheduled shutdown of one incinerator, the second unit was obliged to undergo an unscheduled shutdown.

Conversely, for those emission sources which normally vent to an adsorber or directly to the atmosphere, the emission rate at 300 000 t/a throughput is simply double that at 150 000 t/a. To determine the impact of these different effects as a function of waste throughput, emission rates were estimated for waste throughputs of both 150 000 t/a and 300 000 t/a, as presented in the following sections.

C.2 WASTE TRANSPORTATION

Wastes will arrive at the facility in trucks of varying types and sizes ranging from pick-ups to large multi-axle vehicles. Wastes generally will be containerized in three ways: drums or other types of containers; dump trucks or luggers; and tankers (tank trucks or tank trailers).

For the purposes of estimating emissions, a transportation spill scenario was envisioned whereby a 30 tonne tank truck carrying a load of liquid waste is involved in a collision on the local access road. The collision results in tank rupture and release of the entire contents of the truck. The released liquid spreads across the road surface to form a pool from which the selected surrogate chemical, methylene chloride, is released to the atmosphere. Clearly, different circumstances prevailing at the time of the spill (e.g., nature of the tank rupture, configuration of road surface, shoulder or roadside ditches) will affect the behavior of the liquid pool. As a conservative representation of pool size, the geometry of the pool was assumed to be circular with the center of the circle coincident with the centerline of the road, and the diameter constrained by the width of the paved surface of the road (7.0 m). The emission rate was then based on the equation of Stiver and Mackay (1982):

$$Q = \frac{(K_g)\ (A)\ (P)\ (M)}{(R)\ (T)}$$

where Q = emission rate, g/s

K_g = the gas mass transfer coefficient, m/s

A = area of spill, m^2

P = vapor pressure of the chemical or mixture, Pa

M = molecular weight of compound, g/mol

R = gas constant, $(Pa.m^3)/(mol.K)$

T = temperature, K

This equation was developed for spills of particular chemicals i.e. for a single component system. To apply this approach to the multiple component system found in a waste mixture, the equation was modified to include the mole fraction for each waste component as described in Section C.1.2. The modified emission rate equation then becomes:

$$Q_i = \frac{(Kg_i)(A)(P_i)(x_i)(M_i)}{(R)(T)}$$

where Q_i = emission rate for species i, g/s

Kg_i = the gas mass transfer coefficient for species i, m/s

A = area of the spill, m^2

P_i = vapor pressure for species i, Pa

x_i = mole fraction for species i, dimensionless

M_i = molecular weight of species i, g/mol

R = gas constant, $(Pa.\ m^3)/(mol.K)$

T = temperature, K

Use of this equation requires the calculation of a mass transfer coefficient for the chemical of concern, which can be estimated using the equation of Mackay and Matsugu (1973):

$$Kg = 0.0048\ (U)^{0.78}\ (d)^{-0.11}\ Sc^{-0.67}$$

where Kg = mass transfer coefficient, m/s

U = windspeed, m/s

d = diameter of pool, m

Sc = Schmidt number, dimensionless

Schmidt numbers, which represent the effect of fluid friction on mass transfer, were approximated using the method of Shen (1982) based on molecular weight of the compound. Compounds with molecular weights less than 100 were assigned a $Sc^{-0.67}$ of 0.7, those with molecular weight from 100 to 200 were assigned a $Sc^{-0.67}$ of 0.6, and those with molecular weights greater than 200 were assigned a $Sc^{0.67}$ of 0.5. Values of wind speed and pool diameter were selected for particular spill scenarios.

It is important to recognize that the gas mass transfer coefficient is applicable to the prediction of emission rates of particular chemicals from spills. Mass transfer of a chemical in a complex solution or mixture is a more complicated process, which may involve transport across several phase boundaries. Additionally, transport may be controlled by other materials present in the mixture. However, since these complexities are extremely difficult to quantify for a waste stream and are, in any event, likely to reduce the value of the mass transfer coefficient, it was considered an appropriate and conservative assumption to use the gas mass transfer coefficient for the constituent of concern.

The emission rate calculated in this manner is sensitive to environmental parameters, specifically wind speed, which affects the mass transfer coefficient (Kg) and ambient temperature. For example, the emission rate at 0°C will be approximately three times lower than at 30°C. For the purposes of the risk assessment, emission rates were estimated conservatively by using a temperature of 25°C, considered typical of average summertime temperatures in Southern Ontario. A wind speed of 1 m/s was used to correspond to the atmospheric stability class F subsequently used to model atmospheric transport (see Gartner Lee Associates Limited, 1982 for discussion of atmospheric stability classes).

C.3 BULK WASTE UNLOADING AND TRANSFERS

Waste arriving at the facility will typically be unloaded through below-ground reception basins (unloading tanks). For the initial plant throughput of 150 000 t/a there will be 13 tank truck unloading stations receiving both organic and inorganic wastes; nine for handling inorganic wastes and four for organic wastes. (Additional stations will be added when the plant is expanded to a throughput of 300 000 t/a). A below-ground reception tank equipped with a removable screened cage will be provided for each of the inorganic and two of the organic unloading stations for separating gross solids. For the purpose of calculating the emissions from the basins it was assumed that all wastes (except PCBs) will be unloaded through the reception basins. The reception basins will be equipped with covers or doors and an extraction fan connected to a vent exhaust system. In normal practice, the doors will be opened to 2 m^2 during unloading. Each extraction fan will be designed to maintain a negative pressure in the basin when the doors are closed, and an air velocity of 0.3 m/s in the basin opening when the doors are open. The exhausts from the fans will be ducted to a water scrubber/adsorber system before venting to the atmosphere at a height of 9 metres.

Four scenarios were considered:

- organic waste reception - normal operating conditions (all organics)
- organic waste reception - scrubber failure (methylene chloride as surrogate chemical)
- inorganic waste reception - normal operating conditions (hydrogen cyanide as surrogate chemical)
- inorganic waste reception - scrubber failure (hydrogen cyanide as surrogate chemical)

Emission calculations were performed both for normal operation of the scrubber/adsorber assuming 90% removal of air pollutants, and scrubber/adsorber failure where no pollutant removal was assumed (Monenco, 1985). The calculations are based on a capacity of 150 000 t/a which represents the initial capacity of the facility under normal operation. At the ultimate capacity of 300 000 t/a, emissions under normal operating conditions will be doubled. Emissions under upset conditions were dealt with on a short term basis and will remain unaffected when plant capacity is increased.

C.3.1 ORGANIC WASTE RECEPTION - NORMAL OPERATION

Under normal operating conditions and a facility capacity of 150 000 t/a the emission rate for organic waste unloading and transfer is given by (Monenco, 1985):

Q_i = (molar flow rate of air) (mole fraction of species in gas phase) (adsorber efficiency factor)

$$Q_i = \frac{(V_1 T\ d)}{T_1} \frac{(x_i P_i)}{P_o} M_i\ (1-\eta)\ s\ N_o$$

where

Q_i = emission rate of species i, g/s

V_1 = volumetric flow rate of air into basin per shipment, m^3/s

T_1 = ambient temperature, K

T = standard condition temperature, K

d = molar density of air @ 273K and 760 mm Hg

x_i = mole fraction of species i in waste

P_i = vapor pressure of species i at T_1, mm Hg

P_o = ambient pressure, mm Hg

M_i = molecular weight of species i, g/mol

η = adsorber efficiency

s = percent saturation expressed in fraction

N_o = number of shipments

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- The cross-sectional area of the basin opening will be 2 m^2.
- Air flows in the basin opening at a rate of 0.3 m/s.
- Air leaving the basin will attain 20% saturation with the waste.
- Each shipment will require 15 minutes to unload and three shipments will be received per day for 250 days per year.
- The adsorber efficiency will be 90%.

$V_1 = (2\ m^2)(0.3\ m/s)\dfrac{(15\ min\ 60\ s/min)}{86\ 400\ s} = 0.0063\ m^3/s$
$T_1 = 283K$
$T_s = 273K$
$d = 1/0.0224\ g\ mol/m^3$
$P_o = 760\ mm\ Hg$
$\eta = 0.9$
$s = 0.2$
$N_o = 3$

Therefore, at 150 000 t/a plant capacity, on an average basis, the emission rate of species i (in g/s) is calculated as:

$$Q_i = \frac{(0.0063\ m^3/s)\ (273K)\ (x_iP_i)\ M_i\ (1-0.9)(0.2)(3)}{(283K)\ (0.0224\ m^3/g\ mol)\ (760\ mm\ Hg)}$$

$$= 2.1 \times 10^{-5}\ x_iP_i\ M_i$$

At a peak capacity of 300 000 t/a the emission rate will be doubled so that:

$$Q_i = 4.3 \times 10^{-5}\ x_i\ P_i\ M_i$$

C.3.2 ORGANIC WASTE RECEPTION - SCRUBBER/ADSORBER FAILURE

In the event the scrubber/adsorber fails, i.e., the removal efficiency becomes 0% ($\eta = 0$), and the emission rate due to one shipment becomes:

$$Q_i = \frac{(0.6\ m^3/s)\ (273K)\ (x_i P_i)\ M_i\ (1-0)\ (0.2)\ (1)}{(283K)\ (0.0224\ m^3/g\ mole)\ (760\ mm\ Hg)}$$

$$= 6.8 \times 10^{-3}\ x_i P_i\ M_i \quad g/s$$

Using methylene chloride as the selected surrogate chemical:

$$Q_i = 0.81\ g/s \text{ methylene chloride}$$

C.3.3 INORGANIC WASTE RECEPTION - NORMAL OPERATION

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for inorganic waste unloading and transfer is given by (Monenco, 1985):

$$Q_i = \text{(molar gas flow rate) (adsorber efficiency factor)}$$

$$Q_i = \frac{P_o V}{RT} M\ (1-\eta)$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flow rate of HCN through basin per shipment, m^3/s

R = gas constant, m^3 mm Hg/mol/K

T = standard condition temperature, K

M = molecular weight of HCN

η = adsorber efficiency

Based on the facilty design and operations (Monenco, 1985), the following assumptions were then made for calculation of emissions:

- 50 shipments (800 t/a) of cyanide bearing sludge will be received per year giving a maximum of 1 shipment per day
- the absorber efficiency will be 90%

P_o = 760 mm Hg
V = 0.00114 m^3 of HCN/shipment/day at 273 K
= 0.00114 m^3/86 400 s
= 1.3×10^{-8} m^3/s
R = 0.0624 m^3 mm Hg/mol/K
T = 273 K
M = 27.03 g/mol
η = 0.9

Therefore, assuming a maximum of 1 shipment per day:

$$Q_i = \frac{(760 \text{ mm Hg})(1.3 \times 10^{-8} \text{m}^3/\text{s})(27.03 \text{ g/mol})(1-0.9)}{(0.0624 \text{ m}^3 \text{ mm Hg/mol/K})(273 \text{ K})}$$

$$= 1.6 \times 10^{-6} \text{ g/s hydrogen cyanide}$$

At the peak capacity of 300 000 t/a, the number of shipments becomes 100 shipments per year, so that the average shipment rate will still not exceed one shipment per day and the emission rate will be unchanged on a daily basis.

C.3.4 INORGANIC WASTE RECEPTION - SCRUBBER/ADSORBER FAILURE

In the event the scrubber/adsorber fails, i.e., the removal efficiency becomes 0% (η = 0), the emission rate due to one shipment becomes:

$$Q_i = \frac{(760 \text{ mm Hg})(1.27 \times 10^{-6} \text{ m}^3/\text{s})(27.03 \text{ g/mol})(1-0)}{(0.0624 \text{ m}^3 \text{ mm Hg/mol/K})(273 \text{ K})}$$

$$= 1.52 \times 10^{-3} \text{ g/s}$$

C.3.5 ROUTINE SPILLS

Fugitive emissions may occur while unloading and transferring waste during hose coupling/uncoupling or other related transfer operations. The magnitude of these emissions was based on empirical data developed in a study of hazardous waste transportation spills in the United States (Abkowitz et al., 1984). Data were available from this study for the fraction of cargo lost during various stages in the transportation cycle without any indication of how the spilled chemical would subsequently be partitioned between air, surface water and groundwater.

To estimate the fraction of hazardous waste constituent volatilized to the atmosphere, the same model that was used for characterizing transportation releases in Section C.2 was applied:

$$Q_i = \frac{(Kg_i)(A)(P_i)(x_i)(M_i)}{(R)(T)}$$

where Q_i = emission rate for species, g/s

Kg_i = the gas mass transfer coefficient for species, m/s

A = area of the spill, m^2

P_i = vapor pressure for species, Pa

x_i = mole fraction for species, dimensionless

M_i = molecular weight of species, g/mol

R = gas constant, Pa m^3/mol K

T = temperature, K

The total area of waste generated by routine spills (A) was estimated to be:

$$A = \frac{(W)(fw)}{(b)(\rho)}$$

where A = total spill area, m^2

W = daily quantity of organic waste transported by tanker, tons

fw = fraction of waste released

ρ = average density of wastes, kg/L

b = waste film thickness, m

Based on the facility design and operations, the following assumptions were made to approximate the area of spilled wastes available for volatilization:

- at 150 000 t/a plant capacity, 14 825 t/a organic waste will be transported by tanker (Monenco, 1985).
- the fraction of the waste released at terminal points, i.e. loading and unloading points, will be 8×10^{-6} at the OWMC facility (Abkowitz, M., et al., 1984). For routine spills on-site, the fraction of waste released at unloading points will be 4×10^{-6}.
- the spilled wastes will form a thin film assumed to have a thickness of approximately 0.001 m.
- average waste density is approximately 1.0 kg/L.

It was recognized that the assumptions regarding the thickness of the waste film is subject to considerable uncertainty. This assumption however will likely overestimate emissions since the concrete pad in the unloading area will likely tend to absorb some fraction of spilled liquid. Similarly rainfall events will periodically remove such spillage to the facility drainage system, so that the spill area is conservatively estimated as:

$$A = \frac{(14\ 825\ t/a)(4 \times 10^{-6})(10^{3}\ kg/t)(10^{-3}\ m^{3}/L)}{(250\ d/a)(0.001\ m)(1.0\ kg/L)}$$

$$= 0.24\ m^{2}$$

Since these losses are transportation-related spillages that occur outside the confines of a building without the option for use of pollution control devices, the releases will occur directly to the atmosphere without any removal in an adsorber unit.

Therefore, at 150 000 t/a plant capacity the emission rate for routine spills is given by:

$$Q_i = \frac{(0.24)(Kg_i)(P_i)(M_i)}{(8.314)(283)}$$

$$= (1.02 \times 10^{-4})(Kg_i)(P_i)(M_i)$$

At a peak capcity of 300 000 t/a the emission rate will be doubled to:

$$Q_i = (2.04 \times 10^{-4})(Kg_i)P_i)(M_i)$$

To ensure that this emission rate does not exceed the available quantity of wastes, a mass balance was performed, and an emission rate calculated assuming that the quantity of wastes spilled per day would all be volatilized during the same day. The lower of the two emission rates was used for the purposes of the risk assessment.

C.4 DRUM UNLOADING AND TRANSFER

The drum storage and handling area will be designed to accept wastes arriving in drums up to 200 L in size. The area will contain four drum unloading stations for organic wastes, each consisting of a vacuum receiver, a suction pipe, a vacuum pump and a transfer pump. Drums will be opened by either removing the lid or screwed filling hole plug or by cutting a hole in the top and tranferring the contents to a receiving tank. The emission calculations were based conservatively on complete removal of the lid.

The unloading operation generates fugitive emissions in three ways: initial drawing of the vacuum in the receiver, maintenance of the vacuum while the wastes are pumped from the drums, and displacement of vapor in the tanks as they are filled. Fugitive vapors generated during drum opening will be collected by a ventilation system which exhausts the air through an activated carbon adsorber. Normal emissions are based on an adsorber efficiency of 90%. The emission from the three drum waste transfer operations will be exhausted from the vacuum pumps to a common header which vents to the incinerator. Initially, at 150 000 t/a capacity only one incinerator will be operational. When the incinerator is down, the vacuum pumps will exhaust to an activated carbon adsorber system. These drum transfer emissions were based on 90% efficiency of the carbon adsorption system during the 1760 hours the incinerator is out of service (Monenco, 1985).

At 300 000 t/a capacity there will be two incinerators operational with shut-down time staggered so that at least one incinerator will be functional at all times. Hence, the fugitive emission during drum opening will double but the drum waste transfer emissions will be zero.

C.4.1 DRUM OPENING

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for drum opening is given by (Monenco, 1985); a modified form of the formula is:

$$Q_i = \frac{A \; D_{i,B} \; P \; (P_{i1} - P_{i2}) \; M_i \; (1-\eta)}{R \; T \; Z \; P_{B,m}}$$

where

Q_i = emission rate, g/s

A = liquid surface area of all drums handled daily, cm^2

$D_{i,B}$ = diffusivity of species i in air @ 25°C, cm^2/s

P = ambient pressure, mm Hg

P_{i1} = partial pressure of species i @ gas-liquid interface; $P_{i1} = (P_i)(x_i)$

P_{i2} = partial pressure of species i, 5 cm from surface of liquid

$P_{B,m}$ = logarithmic mean of the partial pressures of gases other than i at the waste-air interface and 5 cm from the surface of liquid

R = gas constant, $(cm^3.mm\ Hg)(g\ mol.K)$

M_i = molecular weight of species i

T = ambient temperature, K

x_i = mole fraction of i in wastes

Z = diffusion distance, cm

η = adsorber efficiency

P_i = vapor pressure of species i

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- The adsorber efficiency is 90%.
- A safety factor of 3 times the calculated diffusion rate was incorporated (Monenco, 1985).
- 10 drums, each of cross-sectional area 2420 cm^2, will be open for 7 h per day.

A = 24200 cm^2
P = 760 mm Hg
P_{i2} = 0 mm Hg

$$P_{B,m} = \frac{(760-0) - (760-x_iP_i)}{\ln \dfrac{(760-0)}{(760-x_iP_i)}} = \frac{x_iP_i}{\ln[760/(760-x_iP_i)]}$$

R = 62400(cm^3.mm Hg)/(g mol.K)
T = 298 K
Z = 5 cm
η = 0.9

Therefore, at 150 000 t/a plant capacity, on a daily average basis:

$$Q_i = \frac{3(24200\text{cm}^2)(D_{i,B})(760\text{ mm Hg})x_iP_iM_i(7\text{h})(1-0.9)\ln[760/(760-x_iP_i)]}{62400(\text{cm}^3.\text{mm Hg})/(\text{g mol.K})\ (298\text{ K})\ (5\text{ cm})\ (24\text{ h})\ (x_iP_i)}$$

$$= 0.0173\ D_{i,B}\ M_i\ \ln\ [760/(760-x_iP_i)]$$

At the peak capacity of 300 000 t/a the emission rate will be doubled so that:

$$Q_i = 0.0346\ D_{i,B}\ M_i\ \ln\ [760/(760-x_iP_i)]$$

C.4.2 DRUM TRANSFER

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate via drum transfer is the summation of the emission rates due to:

- establishment of initial evacuation
- maintenance of vacuum
- displacement of vapor while filling tank

The contribution of each of these emissions was calculated as follows:

a) Establishment of initial evacuation

The emission rate for initial evacuation of the receiving tank is given by (Monenco, 1985):

$$Q_{ai} = \text{(molar flowrate of air)(mole fraction of species in gas phase)(adsorber efficiency factor)}$$

$$= \frac{(P_1 - P_2)\,V}{RT} \; \frac{(x_i P_i)\,M_i\,(1-\eta)}{P_1}$$

where Q_{ai} = emission rate, g/s

P_1 = initial system pressure mm Hg

P_2 = final system pressure, mm Hg

R = gas constant, $(m^3 \cdot mm\ Hg)/(mol \cdot K)$

T = ambient temperature, K

V = volumetric flowrate

P_i = vapor pressure of species i @ T

M_i = molecular weight of i

x_i = mole fraction of i in liquid phase

η = adsorber efficiency

Based on the facility design and operations (Monenco, 1985) , the following assumptions were made for calculation of emissions:

- assume 90% efficiency of adsorber ($\eta = 0.9$)
- volumetric flowrate at 150 000 t/a initial capacity would be 32 m^3/day

$$V = \frac{32\ m^3}{86\ 400\ s} = 3.7 \times 10^{-4}\ m^3/s$$

P_1 = 760 mm Hg
P_2 = 400 mm Hg
R = 0.0624 (m^3.mm Hg)/(mol.K)
T = 298 K

Therefore, at 150 000 t/a plant capacity:

$$Q_{ai} = \frac{(760-400\ \text{mm Hg})(3.7 \times 10^{-4}\ m^3/s)P_i x_i M_i(1-0.9)}{0.0624\ (m^3.\text{mm Hg})(\text{mol.K})\ (298\ K)\ (760\ \text{mm Hg})}$$

$$= 9.42 \times 10^{-7}\ P_i\ x_i\ M_i$$

b) Maintenance of vacuum

The emission rate for maintenance of pressure is given by (Monenco, 1985):

Q_{bi} = (molar flowrate of air) (mole fraction of species in gas phase) (adsorber efficiency factor)

$$Q_{bi} = \frac{(PV)\ (x_i P_i)M_i\ (1-\eta)}{RT\ \ P_o}$$

where

Q_{bi} = emission rate, g/s

P = standard condition pressure, mm Hg

V = volumetric leak rate at standard condition, m^3/s

T = standard condition temperature, K

R = gas constant, $(m^3.mm\ Hg)/(mol.K)$

P_o = system pressure, mm Hg

x_i = mole fraction of species in waste

η = adsorption efficiency

P_i = vapor pressure of species i @ T, mm Hg

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- assume 90% efficiency of adsorber ($\eta = 0.9$)
- assume 2 systems, each leaking @ 2.5 m^3/h for 7 h/d

P_s = 760 mm Hg
V = 2 (2.5 m^3/h) (7 h/d) (1 day/8.64 x 10^4s)
= 4.07 x 10^{-4} m^3/s
T = 298 K
R = 0.0624 $(m^3.mm\ Hg)/(mol.K)$
P = 400 mm Hg

Therefore, at 150 000 t/a plant capacity:

$$Q_{bi} = \frac{(760\ mm\ Hg) \times (4.07 \times 10^{-4}\ m^3/s) P_i\ x_i\ M_i\ (1-0.9)}{0.0624\ (m^3.mm\ Hg)/(mol.K)(298\ K)\ (400\ mm\ Hg)}$$

$$= 4.1 \times 10^{-6}\ P_i\ x_i\ M_i$$

c) Displacement of vapor while filling tank

The emission rate of displaced vapor while filling the tank is given by (Monenco, 1985):

Q_{ci} = (molar flowrate of air) (mole fraction of species in gas phase) (adsorber efficiency factor)

$$= \frac{(P_oV)}{RT} \frac{(P_i\ x_i\ M_i)\ (1-\eta)}{P_o}$$

$$Q_{ci} = \frac{V\ P_i\ x_i\ M_i\ (1-\eta)}{RT}$$

where

Q_{ci} = emission rate, g/s

V = volumetric flowrate, m^3/s

T = system temperature, K

R = gas constant, (m^3.mm Hg)/(mol.K)

P_o = system pressure, mm Hg

x_i = mole fraction of waste species i in waste

η = adsorption efficiency

P_i = vapor pressure of species i @ T

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 90% efficiency of adsorber (η = 0.9)
- 80% of the 2 systems (volume $16m^3$ each) will be evacuated per day

$$V = \frac{(0.8)\ (2 \times 16\ m^3)}{(86400\ s)} = 2.96 \times 10^{-4}\ m^3/s$$

R = 0.0624 (m^3.mm Hg)/(mol.K)
T = 298 K

Therefore, at 150 000 t/a plant capacity:

$$Q_{ci} = \frac{(2.96 \times 10^{-4}\ m^3/s)\ P_i\ x_i\ M_i\ (1-0.9)}{0.0624\ (m^3.mm\ Hg)/(mol.K)\ (298)}$$

$$= 1.59 \times 10^{-6}\ P_i\ x_i\ M_i$$

Hence, summing Q_{ai}, Q_{bi}, and Q_{ci}, the total emission rate via drum transfer at 150 000 t/a plant capacity, is given by:

$$Q_{Ti} = Q_{ai} + Q_{bi} + Q_{ci}$$

$$= 6.63 \times 10^{-6}\ x_i\ P_i\ M_i$$

At 300 000 t/a the vapor will be vented through the incinerators at all times, so that the fugitive emissions from drum transfer will become zero.

C.5 WASTE STORAGE TANKS

The waste storage tanks will be used for storage, blending and pre-treatment of pumpable wastes received from waste generators and from on-site waste sources. Fugitive emissions of working losses will result from the displacement of vapors from the storage tanks as the tanks are being filled.

For purposes of the risk assessment, three scenarios were considered:

- organic waste storage - normal operating conditions (all organics)
- inorganic waste storage - normal operating conditions (hydrogen cyanide as surrogate chemical)
- tank farm fire - accidental release (methylene chloride as surrogate chemical).

At 150 000 t/a capacity, under normal operating conditions, the displaced vapors from the organic waste storage will be vented to the incinerator, except for 1760 hours, when the incinerator is out of operation and the vapors will be vented through an activated carbon adsorber. The emission rates for fugitive emissions were calculated for a day when the incinerator is out of operation assuming an adsorber efficiency of 90%. At 300 000 t/a plant capacity fugitive emissions will be eliminated by the presence of a second incinerator.

In the case of the tank farm fire, the emission rate was based on the entire contents of a tank containing the selected surrogate chemical, methylene chloride (see Appendix B) boiling off as a result of the tank being surrounded by flames.

C.5.1 ORGANIC WASTE STORAGE

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for organic waste storage, working losses is given by (Monenco, 1985):

$$Q_i = \text{(moles of air) (mole fraction of species in gas phase) (adsorber efficiency factor)}$$

$$= \frac{(P_oV)}{RT}\,\frac{(x_iP_iM_i)}{P_o}\,(1-\eta)\,N_o = \frac{V\,x_i\,P_iM_i(1-\eta)N_o}{RT}$$

where

Q_i = emission rate, g/s

P_o = pressure of system, mm Hg

R = gas constant, = 0.0624 (m^3.mm Hg)/(mol.K)

T = temperature of tank, K

V = volume of shipment

x_i = mole fraction of species i in waste

P_i = vapor pressure of species i @ T

M_i = molecular weight of species i

N_o = number of shipments per day

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 100% saturation of air with species i is attained
- volume of shipment is 15 m^3
- 90% efficiency of adsorber ($\eta = 0.9$)
- 5 shipments arrive per day

T = 283 K
V = 15 m^3
N_o = 5 shipments/86400s = 5.79×10^{-5}/s

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(15\ m^3)\ P_i\ x_i\ M_i\ (5.79 \times 10^{-5}/s)\ (1-\eta)}{0.0624\ (m^3.mm\ Hg)/(mol.K)(283\ K)}$$

$$= 4.91 \times 10^{-6}\ x_i P_i M_i$$

At a peak capacity of 300 000 t/a the displaced vapors will be routed to the incinerator feed air at all times, so that the emission rate becomes zero.

C.5.2 INORGANIC WASTE STORAGE

Under normal operating conditions and a plant capacity of 150 000 t/a, the emission rate for inorganic waste storage working losses is given by (Monenco, 1985):

$$Q_i = \frac{P_o V M_{HCN} (1 - \eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flow rate of contaminant, m^3/s at standard conditions

R = gas constant, $(m^3.mm\ Hg)/(mol.K)$

T = standard condition temperature, K

M = molecular weight of HCN

η = adsorber efficiency

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- the volumetric flow rate is 0.00165 m^3/a
- the adsorber efficiency is 90 %

P_o = 760 mm Hg
V = 0.00165 Nm^3 of HCN per year at 273 K
= 0.00165 $Nm^3/(86\ 400)\ (250d)$
= $7.65 \times 10^{-11}\ Nm^3/s$
R = 0.0624 $(m^3.mm\ Hg)/(mol.K)$
T = 273 K
M = 27.03 g/mol
η = 0.9

Therefore,

$$Q_i = \frac{(760\ \text{mm Hg})(7.65 \times 10^{-11}\ \text{Nm}^3/\text{s})(27.03\ \text{g/mol})(1-0.9)}{0.0624\ (\text{m}^3.\text{mm Hg})/(\text{mol.K})(273\ \text{K})}$$

$$= 9.0 \times 10^{-9}\ \text{g/s}$$

At a peak capacity of 300 000 t/a displaced vapors will be doubled so that:

$$Q_i = 1.8 \times 10^{-8}\ \text{g/s}$$

C.5.3 TANK FARM FIRE

Tank farm fire emissions are also based on a conservative scenario developed in earlier work for OWMC in the selection of a buffer zone (Dillon, 1984), which subsequently was adopted by the engineering consultants (Monenco, 1985). In this scenario it is envisioned that a 10m^3 (2500 gallon) tank full of the selected surrogate chemical methylene chloride is exposed to a fire surrounding the tank. The contents of the tank are boiled off and vented above the level of flame effect. Assuming that the tanks are uninsulated, the rate of boil-off, or the emission rate, was estimated by the method of the American Petroleum Institute (API, 1976) as:

$$Q_i = \frac{2652\ (F)(A_w)^{0.82}}{\Delta_H}$$

where

Q_i = emission rate, g/s

F = insulation factor = 1.0 for a bare vessel

A_w = wetted surface area of tank, ft^2

Δ_H = heat of vaporization 139 Btu/lb

The following assumptions were made (Dillon, 1984):

- the wetted surface area of the tank is 176.7 ft^2
- the tank capacity is 2 500 gallons

Therefore,

$$Q_i = \frac{2652\ (1)(176.7\ ft^2)^{0.82}}{(139\ BTU/lb)}$$

$$= 1328\ g/s$$

C.6 PHYSICAL CHEMICAL TREATMENT

The inorganic wastes will undergo preliminary physical chemical treatment which could be a source of fugitive emission. The following unit operations will be used in the treatment:

- dissolved air flotation and emulsion breaking to separate hydrocarbons from aqueous phase.
- oxidation/reduction reactions for the destruction of cyanides and reduction of chromium,
- neutralization/precipitation reactions for heavy metals removal.

Estimates were made for fugitive emissions arising from these sources assuming 150 000 t/a plant capacity. Additionally, emissions resulting from the mixing of incompatible wastes were estimated.

C.6.1 DISSOLVED AIR FLOTATION (DAF) UNIT

The DAF unit will be employed in the separation of suspended oil from aqueous solutions. The air above the liquid is first pressurized thereby increasing the solubility of air in the liquid. The pressure is then quickly released causing nucleation of air bubbles in the aqueous liquid which bubble to the surface. As the air bubbles rise through the waste liquid, globules of hydrocarbon adhere to the bubbles and are carried to the surface. The end result is that liquid hydrocarbon will tend to collect on the surface of the aqueous waste, where, due to the action of the air a layer of froth will be formed. The emission calculation assumes that the air is saturated with the hydrocarbon in the froth. This air is then released directly to the atmosphere.

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for the DAF unit is given by (Monenco, 1985):

$$Q_i = \text{(molar flowrate of air) (mole fraction of species in gas phase)}$$

$$= \frac{(P_s V)}{RT} \frac{(x_i P_i)}{P_o} M_i = \frac{V\ x_i\ P_i\ M_i}{RT}$$

where

Q_i = emission rate, g/s

P = ambient pressure, mm Hg

R = gas constant, 0.0624 (m^3.mm Hg)/(mol.K)

T = standard condition temperature (273 K)

x_i = mole fraction of species i in waste

P_i = vapor pressure of species i @ 25°C

M_i = molecular weight of species i

V = volumetric flowrate of air at standard conditions, m^3/s

P_s = pressure at standard conditions, ~ P_o

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- the unit will be in operation 19 hours per day on an average basis
- the volumetric flowrate is 0.003 Nm^3/min

$$V = (0.003\ Nm^3/min)(min/60\ s)\ \frac{(19\ h)}{(24\ h)} = 3.96 \times 10^{-5}\ m^3/s$$

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(3.96 \times 10^{-5}\ m^3/s)\ x_i\ P_i\ M_i}{0.0624\ (m^3.mm\ Hg)/(mol.K)(273)}$$

$$= 2.3 \times 10^{-6}\ x_i\ P_i\ M_i$$

At the peak capacity of 300 000 t/a the emissions would double to:

$$Q_i = 4.6 \times 10^{-6}\ x_i\ P_i\ M_i$$

C.6.2 EMULSION BREAKING

The oil (hydrocarbon)/water emulsions will be broken by heating. As the emulsion-breaking tank is filled, vapor will be displaced and vented through an adsorber/scrubber system. Air in equilibrium with hydrocarbon/oil will be vented through an adsorber which is assumed to have an efficiency of 90%.

The following emission calculations are based on a 150 000 t/a plant capacity. The estimates would double at the ultimate capacity of 300 000 t/a. Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for the emulsion-breaker is given by (Monenco, 1984):

$$Q_i = \frac{(P_o V)}{RT} \frac{(x_i P_i)}{P_o} M_i(1-\eta) = \frac{V x_i P_i M_i(1-\eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = pressure of system, 760 mm Hg

T = temperature of gas, 343 K

R = gas constant = 0.0624 (m^3.mm Hg)/(mol.K)

x_i = mole fraction of species i in waste

P_i = vapor pressure of species i at T

M_i = molecular weight of species i

V = volume expelled per second

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 2 batches per day each of 25 m^3
- 90% adsorber efficiency (η=0.9)

$$V = 2\ (25\ m^3/d)(d/8.64 \times 10^4\ s)$$

$$= 5.79 \times 10^{-4}\ m^3/s$$

Thus, the emission rate at 150 000 t/a can be calculated as:

$$Q_i = \frac{(5.79 \times 10^{-4}\ m^3/s)\ M_i\ x_i\ P_i\ (1-0.9)}{0.0624(m^3.mm\ Hg)/(mol.K)\ (343\ K)}$$

$$= 2.70 \times 10^{-6}\ x_i\ P_i\ M_i$$

At the peak capacity of 300 000 t/a the emissions would double to:

$$Q_i = 5.4 \times 10^{-6}\ x_i\ P_i\ M_i$$

C.6.3 CYANIDE TREATMENT

The cyanide bearing waste will be destroyed by the addition of an oxidizing agent such as hypochlorite or hydrogen peroxide. Air from the unit will be vented through the scrubber/adsorber. For calculation of emissions under normal operating conditions, it was assumed that the air leaving the unit will be saturated with hydrogen cyanide and vented through a scrubber with an efficiency of 90%.

The situation was also analyzed under upset conditions. Under abnormal conditions, i.e., when the pH control systems fails, the saturation concentration of hydrogen cyanide in equilibrium with the solution increases while the absorber efficiency remains at 90%. It was assumed that the situation would be brought under control after a brief interval of time.

C.6.3.1 Normal Operations

Under normal operating conditions and a facility capacity of 150 000 t/a, the hydrogen cyanide emission rate is given by (Monenco, 1984):

$$Q_i = \frac{P_o V M_{HCN} (1-\eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flowrate at standard condition

R = gas constant = 0.0624 (m^3.mm Hg)/(mol.K)

T = standard condition temperature, 273 K

η = adsorber efficiency

M_{HCN} = molecular weight of HCN, g/mol

Based on facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 200 batches are treated per year
- 1 batch treated per day evolves 0.0011 m^3 of vapor
- scrubber/adsorber efficiency is 90% (η = 0.9)
- M_{HCN} is 27.03 g/mol

P = 760 mm Hg

$$V = \frac{0.0011 \text{ m}^3}{86400 \text{ s}} = 1.273 \times 10^{-8} \text{ m}^3/\text{s}$$

Therefore, at 150 000 t/a:

$$Q_i = \frac{(760 \text{ mm Hg})\ (1.273 \times 10^{-8} \text{ m}^3/\text{s})\ (27.03 \text{ g})\ (1-0.9)}{0.0624\ (\text{m}^3.\text{mm Hg})/(\text{mol.K})\ (273)}$$

$$= 1.5 \times 10^{-6} \text{ g/s}$$

At the peak capacity of 300 000 t/a the emissions would double to 3.1×10^{-6} g/s.

C.6.3.2 Uncontrolled pH

Under abnormal conditions instead of a normal pH of 9.5, a pH reduction to 7.5 would result in a higher HCN vapor concentration in the gas. Vapor emission over a 6 hour period would be:

$$V = \frac{0.053 \text{ m}^3}{6 \times 3600 \text{ s}} = 2.45 \times 10^{-6} \text{ m}^3/\text{s}$$

Therefore, the emission rate would be:

$$Q_i = \frac{(760 \text{ mm Hg})\ (2.45 \times 10^{-6} \text{ m}^3/\text{s})\ (27.03 \text{ g/mol})\ (1-0.9)}{0.0624\ (\text{m}^3.\text{mm Hg})/(\text{mol.K})\ (273 \text{ K})}$$

$$= 3.0 \times 10^{-4} \text{ g/s of hydrogen cyanide}$$

C.6.4 SULFIDE TREATMENT

The removal of heavy metals from waste solutions will be accomplished by the addition of a sodium sulfide solution to a batch of the waste. In the alkaline state, this results in the precipitation of heavy metals as sulfides, and emission of hydrogen sulfide is negligible.

If, during abnormal conditions, the pH is allowed to fall to the acidic state the concentration of gaseous hydrogen sulfide in equilibrium with the waste solution increases. The emission calculation is based on such a failure with saturated air venting through a water scrubber with an efficiency of 90%.

Under abnormal conditions and a facility capacity of 150 000 t/a, the emission rate of hydrogen sulfide is given by (Monenco, 1985):

$$Q_i = \frac{P_o V \, M_{H2S} \, (1-\eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure (760 mm Hg)

R = gas constant = 0.0624 (m^3.mm Hg)/(mol.K)

T = standard condition temperature (273 K)

V = volumetric flowrate of gas at standard condition

η = scrubber/adsorber efficiency

M_{H2S} = molecular weight, 34.08 g/mol

Based on facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions under abnormal pH situation:

- the adsorber efficiency is 90% (η=0.9)
- the volumetric flowrate of gas is 0.013 m^3/hr
- pH falls to 5

$$V = (0.013\ m^3/h)\ (h/3600\ s)$$

$$= 3.61 \times 10^{-6}\ m^3/s$$

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(760\ mm\ Hg)\ (3.61 \times 10^{-6}\ m^3/s)\ (34.08\ g)\ (1-0.9)}{0.0624\ (m^3.mm\ Hg)/(mol.K)\ (273\ K)}$$

$$= 5.49 \times 10^{-4}\ g/s$$

C.6.5 MIXING OF INCOMPATIBLE WASTES

An accidental release considered in the risk assessment was the generation of hydrogen sulfide as a result of mixing of incompatible wastes. The scenario considered was developed in earlier work for OWMC on the selection of a technical buffer zone (M.M. Dillon, 1984).

In this scenario it was envisioned that 4% sulfuric acid was mixed with sulfide contaminated material in the physical/chemical treatment building. The gas is emitted first into the building and then via its ventilation system to the atmosphere. The situation was simulated in the laboratory on a reduced scale by mixing 4% sulfuric acid with zinc sulfide. Based on the laboratory data the emission rate for this scenario over a 30 minute interval was estimated to be 0.462 g/s (Dillon, 1984).

C.7 INCINERATION

An incinerator will be used to destroy organic wastes at high temperatures. Flue gases from the incinerator will be cleaned by a flue gas treatment system consisting of a spray dryer, an electrostatic precipitator, an induced draft fan and a three stage wet gas scrubber. The incinerator operations will result in air pollutants generated from a number of sources:

- organic chemicals in feed,
- acid gases generated by combustion,
- metals present in the waste feed,
- products of incomplete combustion

Estimates were made for these controlled stack emissions and for emissions under upset conditions in the event of scrubber failure.

C.7.1 CONTROLLED STACK EMISSIONS

Estimation of controlled stack emission rates were based on two approaches: a design destruction efficiency in the case of emissions of residual organics, and empirical data for metals, acid gases and dioxins. These approaches are described in the following sections.

C.7.1.1. Incinerator Stack Emissions - Organics

Under normal operating conditions, and a facility capacity of 150 000 t/a, based on 99.99% DRE the emission rate for organics is given by (Monenco, 1985):

$$Q_i = Q_1 f_1 f_2$$

where

Q_i = emission rate of uncombusted chemical, g/s

Q_1 = waste throughput, g/s

f_1 = fraction of waste that represents constituent of concern

f_2 = fraction of waste uncombusted based on 99.99% DRE

Based on the facility design and operating conditions (Monenco, 1985), the following assumptions were made for calculation of emissions:

- DRE of 99.99% implies $f_2 = 10^{-4}$
- annual throughput = 30 000 metric tons (952.2 g/s)

Therefore, at 150 000 t/a plant capacity:

$$Q_i = (952.2)f_1\ (1 \times 10^{-4})$$
$$= 0.0952\ f_1$$

At the peak capacity of 300 000 t/a, two incinerators each operating at 30 000 t/a is envisioned; resulting in an emission rate of:

$$Q_i = 0.19\ f_1$$

C.7.1.2 Incinerator Stack Emission-Metals

The presence of metals in the wastes results in metals emissions to the atmosphere. Many metals evaporate in the flame, either as such, or in the form of volatile oxides, chlorides or other salts. A small percentage of the metals escapes the incinerator emission controls and vents to the atmosphere. Based on comparative facilities and OWMC facility design information the following are estimates of controlled emissions (Q) arising from metals in the feed to an incinerator (ORF, 1985).

TABLE C-3

STACK EMISSION ESTIMATES - METALS

Metal	Emission Rate @ 150 000 t/a	Emission Rate @ 300 000 t/a
Arsenic	4.2×10^{-5} g/s	8.3×10^{-5} g/s
Beryllium	2.8×10^{-7} g/s	5.6×10^{-7} g/s
Cadmium	4.2×10^{-4} g/s	8.3×10^{-4} g/s
Chromium	9.7×10^{-4} g/s	1.9×10^{-3} g/s
Copper	9.0×10^{-4} g/s	1.8×10^{-3} g/s
Lead	4.7×10^{-3} g/s	9.4×10^{-3} g/s
Mercury	3.5×10^{-4} g/s	6.9×10^{-4} g/s
Nickel	4.2×10^{-4} g/s	8.3×10^{-4} g/s
Zinc	4.9×10^{-3} g/s	9.7×10^{-3} g/s

C.7.1.3 Incinerator Stack Emissions-Acid Gases

Emissions of acid gases will be dependent on the waste composition, the incinerator design and operations, and the gaseous and particulate control equipment. The emission (Q) data presented in the following table for plant capacities of 150 000 t/a and 300 000 t/a were determined from data in the literature for comparable facilities and OWMC facility design information (ORF, 1985).

TABLE C-4

STACK EMISSION ESTIMATES - ACID GASES

Acid Gas	Q @ 150 000 t/a	Q @ 300 000 t/a
HCl	0.28 g/s	0.6 g/s
HF	6.9×10^{-3} g/s	1.4×10^{-2} g/s
SO_2	1.04 g/s	2.07 g/s
NO_x	1.04 g/s	2.07 g/s

C.7.1.4 Incinerator Stack Emissions - Products of Incomplete Combustion

Products of incomplete combustion (PICs) are formed as a result of partial oxidation of chemicals in the waste feed due to the combination of molecular fragments outside of the combustion zone. At present there is limited information on the mechanism by which these PICs are formed, and how their formation can be related to waste feed or incinerator operating conditions (ORF, 1985). In the absence of such information, consideration was given to a particular set of possible PICs: the dioxins and furans. Based on comparative facilities and OWMC facility design information the following are estimates of controlled emissions PICs from the incinerator (ORF, 1985).

TABLE C-5

STACK EMISSION ESTIMATES - PICs

PICs	Emission Rate @ 150 000 t/a	Emission Rate @ 300 000 t/a
Dioxins	1.5×10^{-7} g/s	3.1×10^{-7} g/s
Furans	7.1×10^{-7} g/s	1.4×10^{-6} g/s

C.7.2 INCINERATOR SCRUBBER FAILURE

In the event of a flue gas scrubber failure, the emergency power will be designed to resume operations in less than 2 minutes (Monenco, 1985). Gas flow would decrease to 10% of normal in 4 minutes following failure. In the calculation of emission rates it was assumed that at 150 000 t/a facility capacity one incinerator would be in operation and at 300 000 t/a, two incinerators would be operational. Each incinerator has a capacity of about 30 000 t/a. Based on data supplied by Monenco and the above scenario the emission rates for the two minutes of failure will be:

$$Q_i = \text{(Average volumetric flow rate) (Acid gas concentrations)}$$
$$= (V)(C_g)$$

where

Q_i = emission rate, g/s

V = average volumetric flowrate, 5.5 m^3/s

C_g = acid gas concentrations, g/m^3

The emission rates at 150 000 t/a and 300 000 t/a facility capacity are presented in Table C-6. At 300 000 t/a, it was conservatively assumed that power failure would result in scrubber failure for both incincerator units.

TABLE C-6

STACK EMISSION ESTIMATES - SCRUBBER FAILURE

Acid Gases	Acid Gas Concentrations (g/m^3)	Emission Rate @ 150 000 t/a (g/s)	Emission Rate @ 300 000 t/a (g/s)
HCl	12.618	69.0	138.0
SO_2	9.787	53.5	107.0
HF	0.288	1.6	3.1
NO_x	0.285	1.6	3.1

C.8 SUMMARY OF RESULTS

The emission rates for the facility releases described in earlier sections are summarized in Tables C-7, C-8, C-9 and C-10.

Fugitive emissions for organic chemicals are presented in Tables C-7 and C-8 for plant capacities of 150 000 t/a and 300 000 t/a respectively. A comparison of these emissions data shows that the emissions for waste storage and drum transfer, become zero at 300 000 t/a capacity, while the other emissions are each doubled. The net effect of these changes is for the total fugitive emissions to increase slightly at the peak capacity.

TABLE C-7

FUGITIVE EMISSION RATES (g/s)

AT 150 000 t/a

Chemical Constituent	Waste Unload & Transfer	Drum Opening	Drum Transfer	Storage Tanks	DAF Unit	Emulsion Breaking	Routine Spills	Total Fugitive Emissions
Trichloroethylene	2.21E-03	4.36E-04	1.45E-03	5.10E-04	1.35E-05	9.26E-05	1.09E-04	4.82E-03
Chloroform	2.64E-03	4.94E-04	1.62E-03	6.08E-04			4.62E-05	5.40E-03
1,1,1-Trichloroethane	4.74E-04	9.14E-05	3.35E-04	1.09E-04	3.57E-05	3.12E-04	1.85E-05	1.38E-03
Acrylonitrile	1.63E-04	3.84E-05	1.05E-04	3.77E-05			5.54E-06	3.50E-04
Methylene Chloride	2.54E-03	5.34E-04	1.49E-03	5.85E-04			1.91E-05	5.17E-03
Pyridine	7.67E-05	1.88E-05	5.78E-05	1.77E-05			1.55E-05	1.87E-04
Benzene	2.93E-04	6.10E-05	1.91E-04	6.75E-05	5.26E-06	3.51E-05	1.11E-05	6.63E-04
Toluene	3.14E-04	6.53E-05	2.24E-04	7.24E-05			4.39E-05	7.19E-04
Phenol	2.26E-08	7.33E-09	2.51E-08	5.21E-09			5.43E-08	1.15E-07
Formaldehyde	7.81E-03	2.34E-03	3.96E-03	1.80E-03			5.71E-06	1.59E-02
Petroleum Distillates	3.40E-04	6.20E-05	2.38E-04	7.84E-05			3.32E-04	1.05E-03
Tetraethyl Lead	3.76E-10	8.60E-12	2.51E-10	8.66E-11			6.46E-10	1.37E-09
PCB		1.12E-11	8.14E-11	2.68E-11			2.00E-10	3.19E-10
Ethanol	4.20E-03	1.34E-03	3.29E-03	9.68E-03			3.23E-04	1.01E-02
Styrene	3.94E-04	4.40E-05	1.71E-04	9.09E-05			1.55E-04	8.55E-04
Methyl Ethyl Ketone	4.86E-05	1.03E-05	3.35E-05	1.12E-05			1.85E-06	1.05E-04

TABLE C-8

FUGITIVE EMISSION RATES (g/s)

AT 300 000 t/a

Chemical Constituent	Waste Unload & Transfer	Drum Opening	DAF Unit	Emulsion Breaking	Routine Spills	Total Fugitive Emissions
Trichloroethylene	4.43E-03	8.71E-04	2.70E-05	1.85E-04	2.17E-04	5.73E-03
Chloroform	5.27E-03	9.87E-04			9.25E-05	6.35E-03
1,1,1-Trichloroethane	9.47E-04	1.83E-04	8.71E-05	6.24E-04	3.70E-05	1.88E-03
Acrylonitrile	3.27E-04	7.68E-05			1.11E-05	4.15E-04
Methylene Chloride	5.08E-03	1.07E-03			3.81E-05	6.18E-03
Pyridine	1.53E-04	3.77E-05			3.11E-05	2.22E-04
Benzene	5.85E-04	1.22E-04	1.05E-05	7.02E-05	2.23E-05	8.10E-04
Toluene	6.28E-04	1.31E-04			8.78E-05	8.46E-04
Phenol	4.52E-08	1.47E-08			1.09E-07	1.69E-07
Formaldehyde	1.56E-02	4.68E-03			1.14E-05	2.03E-02
Petroleum Distillates	6.81E-04	1.24E-04			6.63E-04	1.47E-03
Tetraethyl Lead	7.51E-10	1.72E-11			1.29E-09	2.06E-09
PCB		2.24E-11			4.00E-10	4.22E-10
Ethanol	8.40E-03	2.68E-03			6.46E-04	1.17E-02
Styrene	7.89E-04	8.80E-05			3.11E-04	3.99E-04
Methyl Ethyl Ketone	$9.72E \times 10^{-5}$	2.06E-05			3.70E-06	1.22E-04

TABLE C-9

INCINERATOR STACK EMISSION ESTIMATES

AT 300 000 t/a

Chemical Constituent	Emission Rate (g/s)	Chemical Constituent	Emission Rate (g/s)
VOLATILE ORGANICS		ACID GASES	
Trichloroethylene	1.88E-03	Sulfur Dioxide	2.07
Perchloroethylene	1.96E-03	Nitrogen Oxides	2.07
Chloroform	0.00E-00	Hydrogen Chloride	0.56
1,1,1-Trichloroethane	3.46E-03	Hydrogen Fluoride	0.014
Hydrazine	6.42E-05		
Acrylonitrile	6.42E-05	METALS	
Methylene Chloride	1.92E-05		
Pyridine	5.39E-04	Lead	9.4E-03
Benzene	3.85E-04	Zinc	9.7E-03
Toluene	1.84E-03	Nickel	8.3E-04
Phenol	9.39E-04	Chromium	1.9E-03
Formaldehyde	2.48E-04	Arsenic	8.3E-05
Petroleum Distillates	1.15E-02	Cadmium	8.3E-04
Tetraethyl Lead	3.20E-07	Mercury	6.9E-04
PCB	2.06E-04	Copper	1.8E-03
Ethanol	1.06E-02	Beryllium	5.6E-07
Styrene	2.68E-03	DIOXINS/FURANS	
Methyl Ethyl Ketone	3.20E-05	Dioxins	3.1E-07
Cresols	2.88E-03	Furans	1.4E-06

TABLE C-10

EMISSION ESTIMATES FOR UPSET SCENARIOS

Operations	Emission Rate @ 150 000 t/a (g/s)	Emission Rate @ 300 000 t/a (g/s)
Transportation Spill		
Methylene Chloride	170.0	170.0
Organic Waste Reception		
Methylene Chloride	8.1E-1	8.1E-1
Inorganic Waste Reception		
Hydrogen Cyanide	1.5E-3	1.5E-3
Tank Farm Fire		
Methylene Chloride	1.3E+3	1.3E+3
Cyanide Treatment		
Hydrogen Cyanide	2.9E-4	2.9E-4
Sulfide Treatment		
Hydrogen Sulfide	5.5E-4	5.5E-4
Mixing of Incompatible Wastes		
Hydrogen Sulfide	4.6E-1	4.6E-1
Incinerator Scrubber Failure		
Hydrogen Chloride	69.0	138.0
Sulfur Dioxide	53.5	107.0
Hydrogen Fluoride	1.6	3.1
Nitrogen Oxides	1.6	3.1

Fugitive emissions of inorganics, for which our analysis was confined to hydrogen cyanide, occur for two cases. For inorganic waste reception, the estimated emissions at 150 000 t/a and 300 000 t/a were 1.6×10^{-6}. These emissions were considered to represent a negligible risk relative to the fugitive emissions and were not considered further in the risk assessment.

Incinerator emissions are presented in Table C-9 for a plant capacity of 300 000 t/a, and include the estimated values for organics, metals, acid gases and dioxins/furans. At a reduced plant capacity of 150 000 t/a these values would all be halved.

Emissions under upset conditions are presented in Table C-10 for the following upset or accident conditions: transportation spill; scrubber failure for organic waste reception; scrubber failure for inorganic waste reception; the tank farm fire; pH excursions during cyanide treatment; pH excursions during sulfide treatment; mixing of incompatible wastes; and incincerator scrubber failure. We note that, with the exception of the incinerator scrubber failure, each of these emission rates is an episodic event unaffected by changes in plant capacity. For the incinerator scrubber failure, since the hypothesized failure mode was based on power failure, both incinerator units would malfunction, so that the emission rate at 300 000 t/a would be double that at 150 000 t/a. Finally, of all the failure modes considered, only the transportation spill, tank farm fire, mixing of incompatible wastes and incinerator scrubber failure were considered in the risk assessment. The other emissions were considered to represent a negligible risk relative to other accident or upset conditions identified.

REFERENCES

Abkowitz, M., Eiger, A., Srinivasan, S. 1984. Assessing the Releases and Costs Associated with Truck Transport of Hazardous Wastes. Prepared for the Office of Solid Waste, USEPA and ICF Inc., Draft Final Report, Ch. 4.

American Petroleum Institute (API). 1976. Recommended Practice for the Design and Installation of Pressure-Relieveing Systems in Refineries. Part 1-Design. API Recommended Practice 520. Fourth Edition.

CRC Handbook of Chemistry and Physics, eds. Weast, R.C., and Astle, M.J. CRC Press, Inc., Florida, 1983.

M.M. Dillon Limited. 1984. Site Selection Process. Phase 3B: Selection of Candidates Sites: Facilities Characteristics and Technical Buffer Zones. Prepared for Ontario Waste Management Corporation.

M.M. Dillon Limited. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Transportation. Prepared for Ontario Waste Management Corporation.

General Electric Co. (GE). 1980. Material Safety Data Sheets. Nielsen, J.M. ed.

Lyman, W. J., Rechl, W.F., Rosenblatt, D.H. 1982. Handbook of Chemical Property Estimation Methods. McGraw Hill Book Company.

Mabey, W.R., Smith, J.H., Podoll, R.T., Johnson, H.L., Mill, T., Chou, T.W., Gates, J., Partridge, I.W., Jaber, H., and Vandenberg, O. 1982. Aquatic Fate Process Data for Organic Priority Pollutants. Prepared by SRI International for U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Monitoring and Data Support Division, Washington, D.C. EPA Contracts 68-01-3867 and 68-03-2981.

Monenco Limited. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Engineering. Prepared for Ontario Waste Management Corporation.

Ontario Research Foundation (ORF). 1985. Emissions Related Studies: Preliminary Estimates of Air Emisssion. In preparation for Ontario Waste Management Corporation. January.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. 2nd ed. Van Nostrand Reinhold Co., New York.

APPENDIX D

DERIVATION OF ENVIRONMENTAL TRANSPORT DATA

CONTENTS

Page

D.1 INTRODUCTION D-1

D.2 ESTIMATION OF ATMOSPHERIC DEPOSITION RATES D-1

D.3 ESTIMATION OF LEAF SURFACE DEPOSITION FACTOR D-4
    D.3.1 Model for Leaf Surface Deposition D-4
    D.3.2 Estimation of Degradation Rate D-6

D.4 ESTIMATION OF UPTAKE FROM SOIL D-10
    D.4.1 Model for Plant Uptake D-10
    D.4.2 Estimation of Loss Terms D-13

D.5 ESTIMATION OF RETENTION IN CATTLE TISSUES AND MILK D-20

D.6 ESTIMATION OF CONCENTRATIONS IN CISTERNS AND PONDS D-27

D.7 SUMMARY OF RESULTS D-28

REFERENCES D-33

LIST OF TABLES

		Page
D.1	Deposition Velocities	D-3
D.2	Leafy Vegetable Crop Yield and Consumption	D-7
D.3	Data Inputs For Derivation of the Plant Surface Degradation Rate Constant	D-11
D.4	Soil to Plant Partition Coefficients	D-14
D.5	Data Inputs for Estimation of the Soil Volatilization Rate Constant	D-17
D.6	Distribution Coefficients for Metals	D-19
D.7	Soil Loss Rate Constants	D-21
D.8	F_B and F_m Values for Metals	D-22
D.9	Octanol - Water Partition Coefficients	D-23
D.10	Food Factors for Cattle	D-24
D.11	Concentrations of Chemicals in Leafy Vegetables	D-29
D.12	Concentrations in Cattle Tissues	D-30
D.13	Concentrations in Milk	D-31
D.14	Cistern and Pond Concentrations	D-32

D.1 INTRODUCTION

This appendix presents the methodology and supporting data for the estimation of environmental fate and transport of chemicals released from the OWMC facility. The appendix provides inputs to the estimation of releases from transportation spills and to the estimation of indirect exposures via consumption of crops and livestock products. The appendix is organized in the following sections:

- Estimation of Atmospheric Deposition Rates
- Estimation of Leaf Surface Deposition Factor
- Estimation of Uptake From Soil
- Estimation of Retention in Cattle Tissues and Milk

D.2 ESTIMATION OF ATMOSPHERIC DEPOSITION RATES

Chemicals in the atmosphere can come into contact with soil and plant surfaces through wet and dry deposition processes. Dry deposition describes the contact of very small particles and gases in the atmosphere due to atmospheric turbulence and molecular motion. Upon contact, the particle or gas can be retained due to effects such as absorption.

The dry deposition rate is defined by Hanna et al. (1982) as:

$$d_{dry} = C\ V_d$$

where d_{dry} = dry deposition rate, $(\mu g/m^2)/s$

C = chemical concentration near the surface, $\mu g/m^3$

V_d = deposition velocity, m/s

The deposition velocities for the chemicals of concern are presented in Table D.1, as provided by Ontario Research Foundation. The wet deposition process describes the contact of chemicals in the atmosphere due to washout by precipitation. In the case of gases, equilibrium partitioning into raindrops is the driving force for removal of the chemical from the atmosphere. It has been suggested that equilibrium is reached between gas and water droplets after a fall of approximately ten metres (Slinn et al., 1981). Such chemical partitioning between water and air is described in a "washout ratio." Although this ratio varies depending upon the nature of the chemical and whether it is in the form of a gas, colloid, or particle, conservative values were assumed for metals and organic chemicals.

Wet deposition can be defined by the following equation (Hanna et al., 1982):

$$d_{wet} = C\ P\ \frac{1}{D}\ W$$

TABLE D.1

DEPOSITION VELOCITIES

CHEMICAL CONSTITUENT	DEPOSITION VELOCITY Vd (m/s)
TRICHLOROETHYLENE	0.01
PERCHLOROETHYLENE	0.01
CHLOROFORM	0.01
1,1,1-TRICHLOROETHANE	0.01
HYDRAZINE	0.01
ACRYLONITRILE	0.01
METHYLENE CHLORIDE	0.01
PYRIDINE	0.01
BENZENE	0.01
TOLUENE	0.01
PHENOL	0.01
FORMALDEHYDE	0.01
PETROLEUM DISTILLATES	0.01
TETRAETHYL LEAD	0.01
PCB	0.005
ETHANOL	0.01
STYRENE	0.01
METHYL ETHYL KETONE	0.01
CRESOLS	0.01
DIOXINS/FURANS	0.0025
LEAD	0.003
ZINC	0.0062
NICKEL	0.0045
CHROMIUM (VI)	0.005
ARSENIC	0.0022
CADMIUM	0.0045
MERCURY	0.01
COPPER	0.005
BERYLLIUM	0.01

Source: Ontario Research Foundation

where d_{wet} = wet deposition rate, $(\mu g/m^2)/s$

P = annual precipitation = 0.9423 m (Environment Canada, 1982)

D = density of air = 1.2×10^9 $\mu g/m^3$

W = washout ratio = 500 for metals and 50 for organic chemicals (Ontario Research Foundation)

C = concentration of chemical in the air, $\mu g/m^3$

The overall deposition rate used to determine the concentration of pollutant in the plant was obtained from the sum of dry and wet deposition processes by the following equation:

$$d = C\,[V_d + P\,\frac{1}{D}\,W]$$

where d = total chemical deposition rate, $(\mu g/m^2)/s$

C = chemical concentration in air, $\mu g/m^3$

V_d = deposition velocity, m/s

P = average annual precipitation, m/s

D = density of air, $\mu g/m^3$

W = washout ratio, unitless

D.3 ESTIMATION OF LEAF SURFACE DEPOSITION FACTOR

D.3.1 MODEL FOR LEAF SURFACE DEPOSITION

Chemicals in the atmosphere can be deposited onto leaf surfaces due to the dry and wet deposition processes described above. Whether this deposition plays a significant role in plant accumulation is not only controlled by the magnitude of this deposition but also by such factors as the fraction intercepted by vegetation, crop yield and various chemical specific surface degradation processes. Thus, the following equation was used to define the leaf surface deposition factor (Holton et al., 1984):

$$A_d = \frac{r[1 - e^{-kt}]}{Y\ k}$$

where A_d = leaf surface deposition factor, $m^2.s/kg$

k = total rate constant for plant leaf degradation processes, s^{-1}

t = time of the growing season, s

Y = yield of the plant (wet weight), kg/m^2

r = interception fraction of plant = 0.39

The extent of surface coverage is determined by the interception fraction of the plant. Since literature references were unavailable, the interception fraction for leafy vegetables was based on a theoretical model developed by Baes et al. (1984). The model generates a range of possible interception fractions given various assumptions of planting practices. Baes et al. considered leafy vegetables such as lettuce, cabbage, greens (collards, kale, spinach), broccoli, mint, celery, cauliflower, green onions, escarole, and brussel sprouts in the analysis. The maximum interception fraction for mature leafy vegetables was found to be 0.79. From this theoretical maximum interception, Baes et al. generated a maximum interception fraction over the time in the field by taking into account the logistic growth characteristics of plants. The theoretical maximum interception fraction was determined to be 0.39. This theoretical maximum was used in this analysis both for leafy vegetables and for grains and grasses consumed by cattle.

For the estimation of indirect exposure, a growing season of five months was assumed. Leafy vegetables were selected for modeling human exposure due to ingestion of locally grown crops because they are considered to accumulate chemicals to a greater degree than non-leafy vegetables. Crop yield was also considered an important factor since high crop yield has the effect of "diluting" the pollutant concentration in the plant by lowering the surface deposition factor. Crop yields were obtained from Ecologistics for the vegetables determined to have the highest potential to bioaccumulate, specifically, asparagus, lettuce, broccoli, cauliflower, and cabbage. It was then conservatively assumed that an individual's dietary consumption of leafy vegetables is made up entirely of these five vegetables, and a weighted average crop yield was developed based on typical consumption levels for each vegetable (see USEPA, 1978). The crop yields and the weighting factors are shown on Table D.2.

D.3.2 ESTIMATION OF DEGRADATION RATE

Chemicals on a leaf surface can undergo degradative processes such as photolysis, can be washed off by precipitation or lost through volatilization. These processes can decrease the amount of chemical deposited on a leaf surface and were expressed in the model as a total rate constant for surface degradation (K).

TABLE D.2

LEAFY VEGETABLE CROP YIELD AND CONSUMPTION

Crop	Yield[1] kg/m^2	Intake[2] lbs/wk/household
Asparagus	0.17	0.14
Broccoli	0.88	0.10
Cabbage	4.09	0.72
Cauliflower	2.09	0.07
Lettuce	2.20	1.28
	Weighted Average = 2.61 kg/m^2	

Source: [1]Ecologistics
[2]USEPA, 1978. Food Factor Tables.

Photolysis

Photolysis describes chemical losses due to transformations caused by the energy of the sun. Organic chemicals are particularly susceptible to such transformations and the photolysis rate constant can be the largest contributor to the total surface loss rate constant, thereby dominating the degradative processes. Of the chemicals selected for modeling, the fate of dioxin could most be affected by photolysis. Dioxin can undergo photolysis under certain conditions (e.g., in the presence of a hydrogen donor). Dioxin formed during incineration, however, would tend to be bound to fly ash. In this state, it is uncertain that photolysis would occur. For these reasons, photolysis was not considered in the analysis for dioxin. Furthermore, photolytic degradation was similarly omitted in an effort to introduce an additional margin of safety in considering the degradation of all organic chemicals selected for modeling.

Weathering

The rate constant for weathering (K_w) describes the loss of a deposited chemical due to wash off by precipitation. A weathering half-life of 15 days was assumed (Willis et al. 1980) for all the chemicals modeled in the risk assessment. This half-life was based on leaf wash-off of the pesticide endrin, a highly hydrophobic chemical which would be expected to adsorb strongly to soil or leaf surfaces. The weathering half-life of 15 days is comparable to the 14 day half-life used by Baes et al. (1984) for the wash-off of metals from a leaf surface. The weathering rate constant is defined by:

$$K_w = \frac{0.693}{t_{1/2}} = 5.34 \times 10^{-7}\ s^{-1}$$

where K_w = weathering rate constant, s^{-1}

$t_{1/2}$ = half-life of chemical on the leaf due to washoff = 15 days

Volatilization

Volatilization of organic chemicals from a leaf surface can represent a significant loss. This loss could occur after dry deposition of the chemical onto the leaf surface, or as leaf surface moisture containing the chemical evaporates. Chemicals would be expected to be found in leaf surface moisture due to partitioning after dry deposition, or due to the washout of wet deposition. Adsorption of chemicals onto the waxy cuticle of a leaf would decrease chemical volatilization losses, thereby providing a more conservative estimate of exposure from surface deposition.

Only organic chemical volatilization was modeled since metals deposited onto leaf surfaces in the form of oxides or chlorides would not be lost through volatilization due to their extremely low vapor pressures. No volatilization was assumed for dioxin due to the fact that dioxin deposited onto a leaf surface would tend to be bound to fly ash, and hence would not be readily lost through volatilization.

An empirically derived soil surface volatilization model developed by Dow Chemical Company was used to estimate volatilization from a leaf surface (Lyman et al., 1982). The model incorporates basic properties such as vapor pressure, water solubility and organic carbon adsorption which are also significant properties in volatilization of chemicals from a leaf surface. Assuming that a leaf surface contains 100% organic carbon, the volatilization rate constant can be estimated by:

$$K_V = 67\,697 \frac{P}{K_{oc} S}$$

where K_V = volatilization rate constant, s^{-1}

P = vapor pressure of the chemical, Pa

S = water solubility of the chemical, mg/L

K_{oc} = soil (organic carbon) adsorption coefficient, $(\mu g/g)/(\mu g/ml)$

67 697 = unit conversion factor

A total rate constant for plant leaf degradation processes based on losses due to weathering and volatilization was then derived:

$$K = K_v + K_w$$

where K = total rate constant for plant surface degradation, s^{-1}

K_v = rate constant for volatilization from the leaf surface, s^{-1}

K_w = rate constant for wash off due to precipitation, s^{-1}

As mentioned previously, metals deposited onto leaf surfaces would not be lost through volatilization. Thus, the total rate constant for plant leaf degradation processes for metals equals the rate constant for wash off due to precipitation. The total rate constants for the organic chemicals of concern and the contributions from volatilization are presented in Table D.3.

D.4 ESTIMATION OF UPTAKE FROM SOIL

D.4.1 MODEL FOR PLANT UPTAKE

Chemicals deposited on the soil surface can leach into the soil solution in the root zone and be taken up by a plant. The extent of plant uptake was estimated by a plant-soil partition coefficient, a parameter that describes how a chemical will ideally distribute itself between a plant and soil given a net concentration in the soil. In addition to uptake by the plant, processes such as downward chemical leaching into soil depths, degradation by soil microorganisms, and volatilization upward through pores in the soil also contribute to the removal of a chemical from the root zone. These processes make the chemical less available for plant uptake.

TABLE D.3

DATA INPUTS FOR DERIVATION OF THE PLANT SURFACE DEGRADATION RATE CONSTANT

CHEMICAL CONSTITUENT	SOLUBILITY AT 25C (mg/l)	VAPOR PRESSURE AT 25C (mm Hg)	Koc	PLANT VOLATIZ. RATE CONSTANT (1/s)	TOTAL PLANT SURFACE DEG. RATE (1/s)
TRICHLOROETHYLENE	1.10E+03	7.38E+01	1.26E+02	2.71E-01	2.71E-01
PERCHLOROETHYLENE	1.50E+02	1.40E+01	3.64E+02	1.31E-01	1.31E-01
CHLOROFORM	9.30E+03	1.93E+02	4.40E+01	2.41E-01	2.41E-01
1,1,1-TRICHLOROETHANE	4.40E+03	1.00E+02	1.52E+02	7.61E-02	7.61E-02
HYDRAZINE	1.00E+06	1.60E+01	4.30E+00	1.89E-03	1.89E-03
ACRYLONITRILE	7.90E+04	1.05E+02	8.50E-01	7.96E-01	7.96E-01
METHYLENE CHLORIDE	1.67E+04	4.33E+02	8.80E+00	1.50E+00	1.50E+00
PYRIDINE	3.00E+08	2.06E+01	2.65E-02	1.32E-03	1.32E-03
BENZENE	1.78E+03	9.45E+01	6.50E+01	4.16E-01	4.16E-01
TOLUENE	5.15E+02	2.82E+01	3.00E+02	9.30E-02	9.30E-02
PHENOL	9.30E+04	5.00E-01	1.42E+01	1.93E-04	1.93E-04
FORMALDEHYDE	6.00E+05	3.83E+03	1.50E-01	2.17E+01	2.17E+01
PETROLEUM DISTILLATES	9.34E-02	4.00E+00	1.60E+04	1.36E+00	1.36E+00
TETRAETHYL LEAD	3.10E+01	1.52E-01	7.30E+03	3.42E-04	3.42E-04
PCB	3.10E-02	7.71E-05	5.30E+05	2.39E-06	2.92E-06
ETHANOL	1.00E+06	5.86E+01	1.60E+01	1.86E-03	1.87E-03
STYRENE	3.00E+02	6.12E+00	1.89E+02	5.49E-02	5.49E-02
METHYL ETHYL KETONE	3.53E+05	1.00E+02	1.00E-02	1.44E+01	1.44E+01
CRESOLS	2.35E+04	1.75E-01	3.54E+01	1.07E-04	1.08E-04
DIOXINS	1.93E-05	1.00E-06	3.30E+06	0.00E+00	5.30E-07
FURANS	1.50E-02	2.00E-06	7.89E+05	0.00E+00	5.30E-07

NOTE: K = kv + Kw where Kw = 5.34E-07

The extent of plant uptake was estimated using the following equation (Holton et al., 1984):

$$Up = \frac{B[1 - e^{-kt}]}{D\ z\ k}$$

where Up = plant uptake factor, $m^2.s/kg$

B = soil-plant partition coefficient, unitless

D = soil density, kg/m^3

z = root zone depth, m

k = total rate constant for soil degradation processes, s^{-1}

t = growing season, s

Soil-plant partition coefficients can be defined as the ratio of the concentration of chemical in the plant to the concentration of the chemical in the soil, obtained under equilibrium or steady state conditions. Such partition coefficients are as yet poorly defined and are not easily extrapolated from a given system due to variations in both soils and plant species. Two sources of information were considered in developing an approach to estimating soil-plant partition coefficients for this risk assessment. The first of these was a food chain risk assessment done by the US Army Medical Bioengineering Research and Development Laboratory (Rosenblatt and Small, 1981) . This work suggests that there may be some correlation between chemical solubility and the soil-plant partition coefficient and used a value for the partition coefficient of 1.0 for substances with solubilities between 0.002 to 10,000 ppm. The solubilities of the chemicals examined in this risk assessment range from 2×10^{-4} ppm to miscible with water, suggesting that the soil-plant partition coefficient of 1.0 would be applicable for the majority of surrogate chemicals.

The second source of data was the microcosm study of Figge, Klahn and Koch (1983) which derives partition coefficients based on the percentage of radioactivity recovered in plants and soil. This work was utilized because the partitioning of a wide variety of chemicals (specifically, perylene, 2,4,5-T, 4-nitrophenol, DDT, DOP, PCP, $HgCl_2$, phenol, 2-nitrophenol, aniline, trichloroethylene, and toluene) between air, soil, plants and percolating water in a microcosm environment was addressed. The maximum partition coefficient derived from this work was 1.8 in the case of phenol. A default value of 2.0 was therefore adopted for all organic chemicals as a conservative approximation.

Baes et al. (1984) have calculated partition coefficients for radionuclides. The soil to plant partition coefficients calculated by Baes were used in the analysis for metals. These values are shown in Table D.4.

D.4.2 ESTIMATION OF LOSS TERMS

The model developed by Holton et al. (1984) addresses soil loss due to biodegradation and leaching. This analysis extends the loss process to include volatilization upward from the root zone through soil pores by the method of Bomberger et al. (1982). Such losses could be significant in removing chemicals of high volatility which do not tend to adsorb onto soil particles. In the case of dioxin bound to fly ash, no volatilization was assumed.

TABLE D.4

SOIL TO PLANT PARTITION COEFFICIENTS

Chemical Constituent	B
Lead	0.045
Zinc	1.5
Nickel	0.06
Chromium	0.0075
Arsenic	0.04
Cadmium	0.55
Mercury	0.9
Copper	0.4
Beryllium	0.01

Source: Baes et al. (1984)

Volatilization

The derivation of a soil volatilization rate constant was applied only to organic chemicals since inorganic chemicals would not be lost through volatilization. The rate constant was based on a half life for volatilization of chemicals from a root zone depth of 15 cm:

$$K_{SV} = \frac{0.693}{t_{1/2}}$$

where: K_{SV} = volatilization rate constant, s^{-1}

$t_{1/2}$ = half-life for soil volatilization, s

The half-life for volatilization from a depth of 15 cm was calculated by the method of Bomberger et al. (1982):

$$t_{1/2} = \frac{L^2}{4.16\, D^A_{soil}}$$

where $t_{1/2}$ = the half-life for diffusion of a chemical from the soil, s^{-1}

L = depth at which the chemical is found; root zone = 15 cm

D^A_{soil} = diffusion coefficient for the chemical in soil air, cm^2/s

The diffusion coefficient for the chemical in soil air was calculated in the following manner (Bomberger et al., 1982):

$$D^A_{soil} = \frac{D^A T_A}{\alpha}$$

where D^A_{soil} = diffusion coefficient for the chemical in soil air, cm^2/s

D^A = diffusion coefficient for the chemical in free air, cm^2/s

T_A = correction for the porosity of the soil

α = correction factor for chemical adsorption on the soil solid and dissolution in the soil water

The correction factors are defined in the following manner:

$$T_A = \frac{0_A^{\ 10/3}}{(0_A + 0_w)^2}$$

where O_A = volume fraction of the soil that is air = 0.26

O_w = volume fraction of the soil that is water = 0.24

and

$$\alpha = O_A + \frac{O_w}{H} + \frac{\rho K_{oc} F_{oc}}{H}$$

where H = Henry's Law constant, dimensionless

ρ = soil bulk density = 1.35 g/ml

K_{oc} = soil adsorption coefficient based on organic carbon

F_{oc} = fraction of organic carbon in the soil = 0.01

O_A = volume fraction of the soil that is air, dimensionless

O_w = volume fraction of the soil that is water, dimensionless

It was assumed that the fraction of organic carbon in the soil would typically be in the order of 1% (Gartner Lee Associates). The Henry's Law constants and diffusion coefficients for the chemicals of concern are presented in Table D.5.

Leaching

Chemicals that are soluble in water will leach from the soil to some degree and tend to migrate vertically downwards from the root zone. The extent of such migration is dependent upon the amount of the precipitation, the soil porosity and the tendency of the chemical to resist movement by adsorption onto organic carbon found in the soil. This tendency is expressed by a partition coefficient (K_{oc}) which describes the distribution of a chemical between soil organic carbon and water. Therefore, soils with a large fraction

TABLE D.5

DATA INPUTS FOR ESTIMATION OF THE SOIL VOLATILIZATION RATE CONSTANT

CHEMICAL CONSTITUENT	DIFF IN AIR (cm^2/s)	HENRY'S LAW CONSTANT (unitless)
TRICHLOROETHYLENE	8.75E-02	3.79E-01
PERCHLOROETHYLENE	7.97E-02	6.38E-01
CHLOROFORM	8.88E-02	1.20E-01
1,1,1-TRICHLOROETHANE	7.94E-02	1.25E+00
HYDRAZINE	1.90E-01	5.40E-07
ACRYLONITRILE	1.06E-01	3.60E-03
METHYLENE CHLORIDE	1.04E-01	8.46E-02
PYRIDINE	9.50E-02	2.90E-07
BENZENE	9.32E-02	2.25E-01
TOLUENE	8.49E-02	2.78E-01
PHENOL	8.50E-02	1.86E-05
FORMALDEHYDE	1.70E-01	1.15E-02
PETROLEUM DISTILLATES	7.57E-02	2.38E+02
TETRAETHYL LEAD	1.00E-02	3.28E+00
PCB	4.00E-02	1.07E-01
ETHANOL	1.20E-01	1.46E-04
STYRENE	7.50E-02	1.87E-01
METHYL ETHYL KETONE	8.94E-02	1.50E-03
CRESOLS	8.10E-02	4.34E-05
DIOXINS	5.00E-02	8.58E-02
FURANS	5.06E-02	2.51E-03

of organic carbon will have a greater tendency to retain certain organic chemicals than soils with a low fraction of organic carbon. As in the calculation of volatilization, it was assumed that the soils in question contain 1% organic carbon (Gartner Lee Associates) in order to calculate a leaching loss rate constant.

The leaching loss rate constant was calculated by the method of Baes et al. (1984) for a clay soil of 1% organic carbon and a volumetric water content of 0.24:

$$k_1 = \frac{P - E}{O_w\, z\, \left[1 + \frac{(\rho\, K_d)}{O_w}\right]}$$

where k_1 = rate constant for leaching loss, s^{-1}

P = annual average total precipitation = 2.991×10^{-6} cm/s (Environment Canada, 1982)

E = annual average evapotranspiration = 1.693×10^{-6} cm/s (Environment Canada, 1982)

O_w = volumetric water content of the soil = 0.24

z = depth of soil layer from which leaching occurs = 15 cm

ρ = soil bulk density = 1.35 g ml^{-1}

K_d = distribution coefficient (ml/g)

For organic chemicals, the distribution coefficient was defined by:

$$K_d = F_{oc} K_{oc}$$

where K_{oc} = soil adsorption coefficient

F_{oc} = fraction of organic carbon in the soil = 0.01

For the inorganic compounds, K_d values derived by Baes et al. were adopted and are shown in Table D.6.

TABLE D.6

DISTRIBUTION COEFFICIENTS FOR METALS

Chemical Constituent	K_d
Lead	900
Zinc	40
Nickel	150
Chromium	850
Arsenic	200
Cadmium	65
Mercury	10
Copper	35
Beryllium	650

Source: Baes et. al. (1984)

Biodegradation

Biodegradation in soils is highly variable. Rate constants were obtained from the literature and where a range of values was indicated, the most conservative number (lowest rate of biodegradation) was selected. In the case of metals and dioxin bound to fly ash, no biodegradation was assumed. The soil volatilization rate constant, the leaching loss rate constant, biodegradation rate constant and the overall soil loss rate constant are shown on Table D.7.

D.5 ESTIMATION OF RETENTION IN CATTLE TISSUES AND MILK

The concentration of chemical found in beef and milk due to leaf surface deposition and plant uptake was estimated by the model of Holton et al. (1984). The model requires the use of cattle dietary assumptions, the concentration of the chemical found in cattle food crops and the fraction of the chemical that is retained in beef and milk from the consumption of exposed food crops:

$$C_B = F_B (Q_B C_g)$$

$$C_m = F_m (Q_m C_g)$$

where C_B = estimated concentration of the contaminant in beef, ug/kg

C_m = estimated concentration of the contaminant in milk, ug/kg

F_B = fraction of the contaminant consumed each day which is transported and retained in beef, d/kg

F_m = fraction of the contaminant consumed each day which is transported and retained in milk, d/kg

Q_B = quantity of non-leafy vegetation (hay, mixed grains, corn silage eaten by beef cattle each day (wet weight), kg/d (Ecologistics)

Q_m = quantity of non-leafy vegetation (hay, mixed grains, corn silage) eaten by milk cows each day (wet weight), kg/d (Ecologistics)

TABLE D.7

SOIL LOSS RATE CONSTANTS

CHEMICAL CONSTITUENT	SOIL VOLATIZ. RATE CONSTANT (1/s)	SOIL LEACHING RATE CONSTANT (1/s)	BIODEG. RATE CONSTANT (1/s)	TOTAL SOIL LOSS RATE CONSTANT (1/s)
TRICHLOROETHYLENE	9.35E-06	4.47E-08	2.80E-11	9.39E-06
PERCHLOROETHYLENE	5.50E-06	1.68E-08	2.80E-11	5.51E-06
CHLOROFORM	7.08E-06	1.04E-07	1.10E-06	8.29E-06
1,1,1-TRICHLOROETHANE	2.18E-05	3.78E-08	4.40E-07	2.23E-05
HYDRAZINE	1.98E-10	2.90E-07	8.30E-07	1.12E-06
ACRYLONITRILE	8.69E-07	3.44E-07	8.30E-10	1.21E-06
METHYLENE CHLORIDE	1.33E-05	2.41E-07	2.30E-06	1.58E-05
PYRIDINE	6.59E-11	3.59E-07	4.50E-05	4.54E-05
BENZENE	1.03E-05	7.76E-08	5.00E-07	1.08E-05
TOLUENE	3.11E-06	2.02E-08	2.80E-08	3.16E-06
PHENOL	2.11E-09	2.01E-07	8.30E-07	1.03E-06
FORMALDEHYDE	4.59E-06	3.57E-07	3.30E-05	3.79E-05
PETROLEUM DISTILLATES	3.72E-05	4.01E-10	1.20E-06	3.84E-05
TETRAETHYL LEAD	1.89E-07	8.78E-10	0.00E+00	1.90E-07
PCB	3.44E-10	1.21E-11	8.30E-10	1.19E-09
ETHANOL	2.21E-08	1.90E-07	3.30E-05	3.32E-05
STYRENE	2.84E-06	3.11E-08	8.30E-10	2.87E-06
METHYL ETHYL KETONE	3.21E-07	3.60E-07	6.10E-06	6.78E-06
CRESOLS	2.82E-09	1.21E-07	2.27E-05	2.28E-05
DIOXINS/FURANS	0.00E+00	1.95E-12	0.00E+00	1.95E-12
	0.00E+00	8.15E-12	0.00E+00	8.15E-12
LEAD		7.12E-11		7.12E-11
ZINC		1.60E-09		1.60E-09
NICKEL		4.27E-10		4.27E-10
CHROMIUM (VI)		7.54E-11		7.54E-11
ARSENIC		3.20E-10		3.20E-10
CADMIUM		9.80E-10		9.60E-09
MERCURY		6.30E-09		6.30E-09
COPPER		1.82E-09		1.82E-09
BERYLLIUM		9.86E-11		9.86E-11

C_g = estimated concentration of contaminant in grains and grasses, ug/kg

The fraction of the chemical that is retained in the beef and milk after the consumption of exposed grain, hay and corn silage by cattle (F_B and F_m) were derived by Baes et al. (1984) for metals. These values were adopted and are shown in Table D.8. For organic chemicals, F_B and F_m were estimated using correlation equations developed by Holton et al. (1984) and based on the work of Kenaga. These equations use the octanol-water partition coefficient (K_{ow}) which describes how a chemical will distribute itself between a water phase and a hydrophobic octanol phase. Octanol partitioning has been used as an estimate of the lipid solubility of a chemical, and hence how it will be accumulated in body fat. Octanol-water partition coefficients for the chemicals of concern are shown in Table D.9.

TABLE D.8

F_B AND F_m VALUES FOR METALS

Chemical Constituent	F_B	F_m
Lead	3×10^{-4}	2.5×10^{-4}
Zinc	1×10^{-1}	1×10^{-2}
Nickel	6×10^{-3}	1×10^{-3}
Chromium	5.5×10^{-3}	1.5×10^{-3}
Arsenic	2×10^{-3}	6×10^{-5}
Cadmium	5.5×10^{-4}	1×10^{-3}
Mercury	2.5×10^{-1}	4.5×10^{-4}
Copper	1×10^{-2}	1.5×10^{-3}
Beryllium	1×10^{-3}	9×10^{-7}

Source: Baes et al. (1984)

TABLE D.9

OCTANOL-WATER PARTITION COEFFICIENTS

CHEMICAL CONSTITUENT	LOG Kow
TRICHLOROETHYLENE	2.42
PERCHLOROETHYLENE	2.6
CHLOROFORM	1.97
1,1,1-TRICHLOROETHANE	2.51
HYDRAZINE	-1.37
ACRYLONITRILE	-0.92
METHYLENE CHLORIDE	1.26
PYRIDINE	0.64
BENZENE	2.13
TOLUENE	2.69
PHENOL	1.46
FORMALDEHYDE	-0.65
PETROLEUM DISTILLATES	0.67
TETRAETHYL LEAD	4.57
PCB	6.04
ETHANOL	-0.32
STYRENE	2.95
METHYL ETHYL KETONE	0.26
CRESOLS	1.98
DIOXINS/FURANS	6.84

In order to estimate F_B and F_m for organic chemicals, the following regression equations were used (Holton et al., 1984):

$$\log F_B = -5.15 + 0.50 (\log K_{ow})$$

$$\log F_m = -6.13 + 0.50 (\log K_{ow})$$

where F_B = fraction of the chemical consumed each day which is transported and retained in beef, d/kg

F_m = fraction of the chemical consumed each day which is transported and retained in milk, d/kg

K_{ow} = octanol/water partition coefficient

The quantity of hay, corn silage and mixed grain consumed by dairy cows and steers per day (Q_g) was provided by Ecologistics and is shown in Table D.10.

TABLE D.10

FOOD FACTORS FOR CATTLE

	Yield kg/m^2	Daily Intake Dairy Cow, kg (wet)	Daily Intake Steer kg (wet)
Hay	0.74	6.8	1.3
Corn silage	3.05	17.1	3.2
Mixed grain	0.30	5.7	7.7
Total		29.6	12.2
		Weighted average = 1.99 kg/m^2	Weighted average = 1.07 kg/m^2

Source: Ecologistics

The concentration of the chemical in the cattle food crops (C_g) was modeled in the same manner as for leafy vegetables:

$$C_g = d[Ad + Up]$$

where
Ad = leaf surface deposition factor, $m^2.s/kg$
Up = soil uptake factor, $m^2.s/kg$
d = chemical deposition rate, $(ug/m^2)/s$

The leaf deposition factor is defined by Holton et al. (1984) as:

$$Ad = \frac{r\,[1 - e^{-kt}]}{Y\,k}$$

where
Ad = leaf surface deposition factor, m^2-s/kg
k = total rate constant for plant leaf degradation processes, s^{-1}
t = time of the growing season, s
Y = yield of the plant (wet weight), kg/m^2
r = interception fraction of the plant, unitless

As for leafy vegetables, a weighted average for crop yield was determined based on consumption of grains and grasses by steers and dairy cows. Due to differences in grain and grass consumption, separate crop yield factors were calculated for dairy cows (1.99 kg/m^2) and for steers (1.07 kg/m^2).

The soil uptake factor is defined by Holton et al. (1984) as:

$$Up = \frac{B\,[1 - e^{-kt}]}{Dzk}$$

where Up = plant uptake factor, m^2s/kg

B = soil-plant partition coefficient, unitless

D = soil density, kg/m^3

z = root zone depth, m

k = total rate constant for soil degradation processes, s^{-1}

t = growing season, s

In the use of soil-plant partition coefficients (B) for grains and grasses, a factor considered was the distribution of chemicals in the plant. Studies by Baes et al. (1984) on radionuclides suggest that only a small fraction of the chemical taken up in the leafy part of the plant may be incorporated into the reproductive part of the plant, i.e., grain. Thus, for grains, Baes suggests that the soil-plant partition coefficient for metals in grains could be one tenth of that predicted for leafy portions of the plant.

It is unclear, however, whether this relationship can reasonably be applied to organic chemicals. Given this uncertainty and to be consistent with the conservative assumptions used throughout the risk assessment, no allowance for reduced uptake by grains was made in estimating chemical concentrations in beef and milk, either for metals or organic chemicals.

D.6 ESTIMATION OF CONCENTRATIONS IN PONDS AND CISTERNS

Chemical concentrations in ponds and cistern water were conceptualized as being controlled by two different processes depending upon whether gaseous or particulate emissions were being considered. For gaseous pollutants, Henry's Law describes how a chemical will distribute itself between air and water under ideal conditions (Lyman et al., 1982):

$$C_A = (C_s)(H)$$

where C_A = equilibrium air concentration of the chemical above the dilute aqueous solution, g/cm^3

C_s = equilibrium aqueous concentration (dilute) of the chemical, g/cm^3

H = Henry's Law constant, dimensionless

For chemicals emitted in particulate form or associated with particulates, i.e., incinerator fly ash, Henry's Law cannot be used to estimate the concentrations in pond and cistern water. For example, metals emitted from incinerators will not remain as gas, but will quickly adsorb to fly ash or exist as solid oxides. For particulate emissions, therefore, concentrations were estimated by the mass of material deposited into a volume of water, and subsequently released from the particles by desorption.

Ponds in the vicinity of the candidate sites are typically 0.6 m to 7.6 m in depth (Gartner Lee Associates). Neglecting any dilution afforded by the initial contents of the pond, the long term concentration in such a pond can be conservatively estimated as:

$$C_m = \frac{(d)(k_s)}{P-E}$$

where C_m = concentration of metal in the pond, g/m^3

d = deposition rate, $g/m^2.a$

P = average annual precipitation, m/a

E = average annual evapotranspiration, m/a

k_s = sediment water partition coefficient

The particulate deposition approach was used in estimating chemical concentrations for all metals emitted from the incinerator. As a conservative assumption, it was also assumed that the sediment-water partition coefficient, i.e., the ratio of the mass of chemical in solution to the mass of chemical adsorbed on the sediment, would be one. For organic chemicals, the Henry's Law approach was applied with the exception of dioxins. Since dioxins may be strongly adsorbed to fly ash emitted from the incinerator, both the Henry's Law and particulate deposition approaches were considered. A sediment-soil partition coefficient of 0.09 was adopted as the lowest value found in reviewing the literature (Matsumura and Ward, 1976). Using both methods the particulate deposition approach was found to result in the highest water concentrations. Consistent with the conservative approach used throughout the generic risk assessment, this higher value was adopted for assessing exposure.

D.7 SUMMARY OF RESULTS

The chemical concentrations predicted in leafy vegetables, cattle tissues and milk, and ponds and cisterns are presented in Tables D.11, D.12, D.13 and D.14, respectively.

TABLE D.11

CONCENTRATIONS OF CHEMICALS IN LEAFY VEGETABLES

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)
TRICHLOROETHYLENE	3.77E-01	1.32E+00	7.40E-04	4.44E-03
PERCHLOROETHYLENE	0.00E+00	0.00E+00	1.31E-03	7.88E-03
CHLOROFORM	4.40E-01	1.53E+00	0.00E+00	0.00E+00
1,1,1-TRICHLOROETHANE	3.84E-02	1.31E-01	5.75E-04	3.45E-03
HYDRAZINE	0.00E+00	0.00E+00	2.12E-04	1.27E-03
ACRYLONITRILE	2.28E-01	8.00E-01	1.95E-04	1.17E-03
METHYLENE CHLORIDE	2.32E-01	8.08E-01	4.48E-06	2.69E-05
PYRIDINE	5.28E-03	1.88E-02	5.35E-05	3.21E-04
BENZENE	4.43E-02	1.54E-01	1.31E-04	7.86E-04
TOLUENE	2.06E-01	7.30E-01	2.15E-03	1.29E-02
PHENOL	3.06E-04	1.12E-03	3.47E-03	2.08E-02
FORMALDEHYDE	3.79E-01	1.34E+00	2.41E-05	1.44E-04
PETROLEUM DISTILLATES	5.28E-02	1.92E-01	1.10E-03	6.62E-03
TETRAETHYL LEAD	1.60E-05	5.81E-05	5.70E-06	3.42E-05
PCB	8.95E-06	3.29E-05	6.68E-03	4.01E-02
ETHANOL	3.25E-01	1.16E+00	1.31E-03	7.86E-03
STYRENE	3.89E-01	1.39E+00	3.45E-03	2.07E-02
METHYL ETHYL KETONE	1.15E-02	4.02E-02	1.74E-05	1.04E-04
CRESOLS	0.00E+00	0.00E+00	1.10E-03	6.59E-03
LEAD	0.00E+00	0.00E+00	8.22E-04	6.85E-03
ZINC	0.00E+00	0.00E+00	1.70E-03	1.41E-02
NICKEL	0.00E+00	0.00E+00	7.80E-06	6.50E-05
CHROMIUM (VI)	0.00E+00	0.00E+00	8.76E-05	7.30E-04
ARSENIC	0.00E+00	0.00E+00	7.00E-06	5.83E-05
CADMIUM	0.00E+00	0.00E+00	9.79E-05	8.16E-04
MERCURY	0.00E+00	0.00E+00	1.13E-04	9.41E-04
COPPER	0.00E+00	0.00E+00	2.03E-04	1.69E-03
BERYLLIUM	0.00E+00	0.00E+00	6.20E-08	5.17E-07
DIOXINS	0.00E+00	0.00E+00	1.06E-05	6.38E-05
FURANS	0.00E+00	0.00E+00	4.92E-05	2.95E-04

TABLE D.12

CONCENTRATIONS IN CATTLE TISSUES

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)
TRICHLOROETHYLENE	5.24E-04	1.83E-03	1.03E-06	6.18E-06
PERCHLOROETHYLENE	0.00E+00	0.00E+00	2.25E-06	1.35E-05
CHLOROFORM	3.64E-04	1.27E-03	0.00E+00	0.00E+00
1,1,1-TRICHLOROETHANE	5.94E-05	2.03E-04	8.89E-07	5.33E-06
HYDRAZINE	0.00E+00	0.00E+00	3.78E-09	2.27E-03
ACRYLONITRILE	6.76E-06	2.38E-05	5.80E-09	3.48E-03
METHYLENE CHLORIDE	8.47E-05	2.95E-04	1.64E-09	9.84E-09
PYRIDINE	1.19E-06	4.25E-06	1.21E-08	7.25E-08
BENZENE	4.41E-05	1.54E-04	1.30E-07	7.83E-07
TOLUENE	3.91E-04	1.38E-03	4.08E-06	2.45E-05
PHENOL	1.48E-07	5.40E-07	1.67E-06	1.00E-05
FORMALDEHYDE	1.54E-05	5.43E-05	9.76E-10	5.86E-09
PETROLEUM DISTILLATES	9.79E-06	3.56E-05	2.05E-07	1.23E-06
TETRAETHYL LEAD	2.65E-07	9.65E-07	9.47E-08	5.68E-07
PCB	9.69E-07	3.56E-06	7.24E-04	4.34E-03
ETHANOL	2.21E-05	7.86E-05	8.91E-08	5.35E-07
STYRENE	9.95E-04	3.56E-03	8.83E-06	5.30E-05
METHYL ETHYL KETONE	1.33E-06	4.64E-06	2.01E-09	1.21E-08
CRESOLS	0.00E+00	0.00E+00	1.68E-06	1.01E-05
LEAD	0.00E+00	0.00E+00	5.16E-03	4.30E-02
ZINC	0.00E+00	0.00E+00	2.66E+00	2.22E+01
NICKEL	0.00E+00	0.00E+00	9.75E-04	8.12E-03
CHROMIUM (VI)	0.00E+00	0.00E+00	1.02E-02	8.50E-02
ARSENIC	0.00E+00	0.00E+00	2.93E-04	2.44E-03
CADMIUM	0.00E+00	0.00E+00	9.89E-04	8.25E-03
MERCURY	0.00E+00	0.00E+00	4.87E-01	4.06E+00
COPPER	0.00E+00	0.00E+00	3.86E-02	3.21E-01
BERYLLIUM	0.00E+00	0.00E+00	1.31E-06	1.09E-05
DIOXINS	0.00E+00	0.00E+00	4.05E-06	2.43E-05
FURANS	0.00E+00	0.00E+00	1.01E-05	6.04E-05

TABLE D.13

CONCENTRATIONS IN MILK

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)
TRICHLOROETHYLENE	1.34E-04	4.69E-04	2.64E-07	1.58E-06
PERCHLOROETHYLENE	0.00E+00	0.00E+00	5.75E-07	3.45E-06
CHLOROFORM	9.32E-05	3.24E-04	0.00E+00	0.00E+00
1,1,1-TRICHLOROETHANE	1.52E-05	5.18E-05	2.27E-07	1.36E-06
HYDRAZINE	0.00E+00	0.00E+00	9.62E-10	5.77E-09
ACRYLONITRILE	1.73E-06	6.09E-06	1.48E-09	8.90E-09
METHYLENE CHLORIDE	2.17E-05	7.56E-05	4.20E-10	2.52E-09
PYRIDINE	2.55E-07	9.10E-07	2.59E-09	1.55E-08
BENZENE	1.13E-05	3.93E-05	3.34E-08	2.00E-07
TOLUENE	1.00E-04	3.54E-04	1.04E-06	6.27E-06
PHENOL	3.65E-08	1.33E-07	4.13E-07	2.48E-06
FORMALDEHYDE	3.94E-06	1.39E-05	2.50E-10	1.50E-09
PETROLEUM DISTILLATES	2.51E-06	9.10E-06	5.24E-08	3.14E-07
TETRAETHYL LEAD	6.75E-08	2.46E-07	2.41E-08	1.45E-07
PCB	2.14E-07	7.87E-07	1.60E-04	9.62E-04
ETHANOL	5.08E-06	1.81E-05	2.05E-08	1.23E-07
STYRENE	2.55E-04	9.12E-04	2.26E-06	1.36E-05
METHYL ETHYL KETONE	3.39E-07	1.19E-06	5.15E-10	3.09E-09
CRESOLS	0.00E+00	0.00E+00	2.76E-07	1.66E-06
LEAD	0.00E+00	0.00E+00	5.66E-03	4.72E-02
ZINC	0.00E+00	0.00E+00	4.22E-01	3.52E+00
NICKEL	0.00E+00	0.00E+00	2.14E-04	1.79E-03
CHROMIUM (VI)	0.00E+00	0.00E+00	3.64E-03	3.03E-02
ARSENIC	0.00E+00	0.00E+00	1.16E-05	9.64E-05
CADMIUM	0.00E+00	0.00E+00	2.57E-03	2.14E-02
MERCURY	0.00E+00	0.00E+00	1.30E-03	1.09E-02
COPPER	0.00E+00	0.00E+00	8.08E-03	6.73E-02
BERYLLIUM	0.00E+00	0.00E+00	1.54E-09	1.29E-08
DIOXINS	0.00E+00	0.00E+00	7.01E-07	4.21E-06
FURANS	0.00E+00	0.00E+00	1.74E-06	1.05E-05

TABLE D.14

CISTERN AND POND CONCENTRATIONS

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (g/m^3)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (g/m^3)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (g/m^3)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (g/m^3)
TRICHLOROETHYLENE	9.11E-08	4.43E-07	1.79E-10	1.49E-09
PERCHLOROETHYLENE	0.00E+00	0.00E+00	1.11E-10	9.22E-10
CHLOROFORM	2.96E-07	1.43E-06	0.00E+00	0.00E+00
1,1,1-TRICHLOROETHANE	6.67E-09	3.16E-08	9.98E-11	8.32E-10
HYDRAZINE	0.00E+00	0.00E+00	4.28E-06	3.56E-05
ACRYLONITRILE	7.49E-07	3.65E-06	6.42E-10	5.35E-09
METHYLENE CHLORIDE	4.23E-07	2.05E-06	8.18E-12	6.82E-11
PYRIDINE	6.59E-03	3.27E-02	6.69E-05	5.57E-04
BENZENE	2.08E-08	1.01E-07	6.15E-11	5.13E-10
TOLUENE	2.28E-08	1.12E-07	2.38E-10	1.99E-09
PHENOL	1.61E-07	8.15E-07	1.82E-06	1.51E-05
FORMALDEHYDE	1.22E-05	5.99E-05	7.75E-10	6.46E-09
PETROLEUM DISTILLATES	8.32E-11	4.20E-10	1.74E-12	1.45E-11
TETRAETHYL LEAD	9.82E-15	4.97E-14	3.51E-15	2.93E-14
PCB	9.26E-14	4.73E-13	6.92E-11	5.77E-10
ETHANOL	6.47E-04	3.20E-03	2.61E-06	2.18E-05
STYRENE	5.82E-08	2.90E-07	5.17E-10	4.31E-09
METHYL ETHYL KETONE	5.06E-07	2.46E-06	7.68E-10	6.40E-09
CRESOLS	0.00E+00	0.00E+00	2.38E-06	1.99E-05
LEAD	0.00E+00	0.00E+00	3.75E-04	3.13E-03
ZINC	0.00E+00	0.00E+00	4.40E-04	3.67E-03
NICKEL	0.00E+00	0.00E+00	3.53E-06	2.94E-05
CHROMIUM (VI)	0.00E+00	0.00E+00	4.08E-05	3.40E-04
ARSENIC	0.00E+00	0.00E+00	3.20E-06	2.66E-05
CADMIUM	0.00E+00	0.00E+00	3.53E-05	2.94E-04
MERCURY	0.00E+00	0.00E+00	3.60E-05	3.00E-04
COPPER	0.00E+00	0.00E+00	7.80E-05	6.50E-04
BERYLLIUM	0.00E+00	0.00E+00	2.88E-08	2.40E-07
DIOXINS	0.00E+00	0.00E+00	2.14E-10	1.78E-09
FURANS	0.00E+00	0.00E+00	9.90E-10	8.25E-09

REFERENCES

Baes, G.F., Sharp, R.D., Sjoreen, A.L. and Shaw, R.W. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides in Agriculture. Oak Ridge National Laboratory, Oak Ridge, TN (ORNL-5786).

Bomberger, D.C., Gwinn, J.L., Mabey, W.R., Tuse, D., and Tsong Wen Chou. 1982. Environmental Fate and Transport at the Terrestrial-Atmospheric Interface. Symposium on Models for Predicting Fate of Chemicals in the Environment. American Chemical Society, Division of Pesticides Chemistry, 184th National Meeting, Kansas City, MO.

Environment Canada, 1982. Canadian Climate Normals. Volume 3 - Precipitation: 1951 - 1980.

Hanna, S.R., Briggs, G.A., and Hosker, R.P. 1982. Handbook on Atmospheric Diffusion. National Turbulence and Diffusion Laboratory, National Oceanic and Atmospheric Administration. DOE/TIC-11223.

Holton, G.A., Travis, C.C., Etnier, E.L., O'Donnell, F.R., Hetrick, D.M. and Dixon, E. 1984. Multiple-Pathways Screening-Level Assessment of a Hazardous Waste Incineration Facility. Oak Ridge National Laboratory, Oak Ridge, TN (ORNL/TM-8652).

Lyman, W.J., Reehl, W.F. and Rosenblatt, D.H. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York.

Mackay D., and Matsugu, R.S. 1973. Evaporation Rates of Liquid Hydrocarbon Spills on Land and Water. Canadian Journal of Chemical Engineering. 51:434, August.

Matsumura and Ward. 1976. As cited in Dioxins. Esposito, M.P., Tiernan, T.D. and Dryden, F.E. November 1980.

Monenco Limited. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Engineering. Prepared for Ontario Waste Management Corporation.

Rosenblatt, D.H. and Small, M.J. 1981. Preliminary Pollutant Limit Values for Alabama Army Munition Plant. U.S. Army Medical Bioengineering Research and Development Laboratory.

Shen, T.T. 1982. A Simplified Method of Hazardous Emissions from Waste Lagoons. Presented at the 75th Annual Meeting of the Air Pollution Control Association, New Orleans, June 20-25.

Slinn, S.A. and Slinn, W.G.N. 1981. Modeling of Atmospheric Particulate Deposition to Natural Waters. As cited in Environmental Exposure from Chemicals - Volume I. CRC Press.

Stiver, W. and Mackay, D. 1982. Spill Technology Newsletter, January/February 1982. As cited in Technical Information for Problem Spills. Draft Introduction Manual.

Willis, G.H., Spencer, W.S. and McDowell, L.L. 1980. The Interception of Applied Pesticides by Foliage and their Persistence and Washoff Potential. A Field Scale Model for Chemical Runoff and Erosion from Agricultural Management Systems. USDA Conservation Research Report No. 26.

United States Environmental Protection Agency. 1978. Update of Food Factor Tables. Memo from R. D. Schmitt, Ph. D., Toxicology Branch to Acting Chief, Toxicology Branch. May 1, 1978.

APPENDIX E

RELATIVE RISKS FOR TRANSPORTATION OF SPECIAL WASTES, REAGENTS AND ADDITIVES, AND INTER-SITE TRANSFERS

CONTENTS

Page

E.1 INTRODUCTION E-1

E.2 METHODOLOGY E-1
Wastes E-4
Reagents and Additives E-7
Inter-Site Transfers E-11

E.3 RESULTS AND ANALYSIS E-12

REFERENCES E-14

LIST OF TABLES

Page

E-1 Special Wastes E-5

E-2 Reagants and Additives E-8

E-3 Inter-Site Transfers E-12

E.1 INTRODUCTION

Direct exposure to toxic pollutants via the air pathway can occur as a result of a spill on the local access route if a spill occurs in proximity to a neighboring residence. Such spills can occur in the transportation of three groups of commodities associated with the OWMC facility: 1) special wastes transported to the facility; 2) reagents and additives transported to the facility; and 3) treatment residues transferred between units in the event that the components of the facility are located at more than one site. In order to determine the relative risks associated with the transportation of these three commodities, a comparative risk assessment was performed. The risk assessment considered a spill event on the local or regional access route resulting in the release of the material and subsequent air dispersion leading to human exposure by inhalation.

E.2 METHODOLOGY

The comparative risk assessment considered both the consequence or severity of the commodity being spilled and the potential frequency of that event occurring. The severity index was based on the inherent hazard of the commodity group being transported and the potential for a toxic pollutant to be released to the atmosphere, in either gaseous or particulate form. The severity index was defined for each commodity in the group as:

$$\text{Severity Index} = \text{Acute Toxicity} \times \text{Release Potential}$$

where Acute Toxicity = LD_{50}

Release Potential = Emission rate

Acute toxicity represents the potential for acute health effects on a human receptor. As a relative measure of the severity of such effects, acute toxicity was evaluated using LD_{50} values from animal studies (e.g., the lethal dose of a chemical needed to kill half the exposed population for the most sensitive mammalian species on which it has been tested). When LD_{50} values were unavailable, acute toxicity was derived from the Threshold Limit Value (TLV).

Since each of the three commodity groups contained several commodities of different hazards, e.g., the inter-site transfers includes incinerator ash and oily wastes, a quantity-weighted average for each of the commodity groups was calculated as:

$$\text{Overall Severity Index} = \frac{\sum (SI_i)(Q_i)}{\sum Q_i}$$

where SI_i = severity index for each commodity i

Q_i = quantity of that commodity

In addition to the severity index, the frequency of a spill of the commodity group being transported was assessed based on the quantity of the commodity being transported. It was assumed that commodity groups transported in large quantities would have a greater potential for a spill occurring. Information on the commodities was based on the data base developed by Canviro (1985) to estimate the types and quantities of special wastes generated in Ontario and on the Phase 4A Engineering Report (Monenco, 1985). Estimates are based on the 300 000 t/a ultimate plant throughout.

In employing this methodology, several assumptions were made about the configuration of the spill and the environmental conditions prevailing at the time of the spill, notably wind speed and ambient temperature. In general, average climatic conditions were used for estimating the release potential of volatile and particulate commodities rather than worst case meteorology for comparison of the risk pathways. These assumptions are summarized below:

Spill Area -- The emission rate is directly proportional to the area of the spill. The spill area is assumed to remain constant over the period of concern and is defined by a circle of diameter equal to the width of the paved road surface. Based on information provided by M.M. Dillon Limited, a pavement width of 7.0 m was selected to define the spill area. In the case of solid materials, the spill was assumed to take the shape of a cone with a diameter of 7.0 m and a height of 1.5 m.

Ambient Temperature -- Seasonal temperature differences will affect the emission rate for volatile organics, since the vapor pressure of a chemical increases with increasing temperature. Spills occurring in the summer months will thus result in a higher emission rate than spills in the winter. A temperature of 10^{0}C was selected for this evaluation as being a typical average annual temperature for Southern Ontario.

Wind Speed -- The emission rate will be affected by changes in wind speed, i.e., the greater the wind speed the greater the emission rate. In the spill scenario considered here, a wind speed of 3 m/s was selected, corresponding to atmospheric stability class D. Stability class D describes a neutral condition and is the most common situation encountered.

The methodology for derivation of the severity index is discussed separately for wastes, reagents and inter-site transfers in the following sections.

WASTES

Since the waste streams accepted at the OWMC facility may contain many hazardous constituents, surrogate chemicals were chosen that represent the most hazardous waste streams to be transported to the facility. The most hazardous waste streams were then used to represent the waste classes. Attention here was focused on those waste classes which were considered most likely to contain significant concentrations of volatile organics. The total quantity of waste containing volatile organics is 59 410 t/a. The remaining 240 590 t/a projected for the 300 000 t/a facility would contain primarily inorganic constituents. The surrogate selection process is addressed in detail in Appendix B of this report. The surrogate chemicals and the quantities of the waste classes they represent are summarized in Table E-1. The acute toxicity was evaluated using LD_{50} values determined for the most sensitive animal species tested.

The release potential was defined as the emission rate of the surrogate chemical from the spill. For volatile organic chemicals, the emission rate was estimated using the following equation for a pure component (Stiver and Mackay, 1982):

$$Q_i = \frac{(Kg)(A)(P_i)(M_i)}{(R)(T)}$$

TABLE E-1

SPECIAL WASTES

Surrogate Chemical	UCD Category	Waste Quantity (t/a)
Class 1: Organic Sludges and Still Bottoms		
Methylene chloride	247	600
Petroleum distillates	248	2400
Phenol	257	2000
Styrene	261	6000
Acrylonitrile	263	4000
Trichloroethylene	446	2000
Formaldehyde	523	2000
Class 2: Solvents and Organic Solutions		
Methylene chloride	211	1400
Trichloroethylene	212	200
Methylene chloride	213	1000
Petroleum distillates	214	1200
Toluene	215	600
Toluene	216	600
Ethanol	217	2000
Acrylonitrile	224	400
Class 3: Oils and Greases		
Petroleum distillates	281	2000
Class 4: Oil and Water Mixtures		
Trichloroethylene and Benzene	283	8000
Class 5: Organic and Oily Residuals		
Petroleum distillates and ethanol	264	20
Phenol	286	600
Petroleum distillates	289	6000
Tetraethyl lead sludge	447	10
Class 7: Miscellaneous Chemicals and Products		
Petroleum distillates and ethanol	243	400
Formaldehyde	245	1000
Ethanol	416	400
Pyridine	513	400
Class 8: Paint and Organic Residuals		
Methylene chloride	254	1000
Formaldehyde	262	1000
Petroleum distillates	454	4000
Petroleum distillates	455	1000

TABLE E-1 (Continued)

SPECIAL WASTES

	Surrogate Chemical	UCD Category	Waste Quantity (t/a)
Class 9:	Aqueous Solution with Organics		
	Formaldehyde	225	3000
	Toluene	227	1000
Class 12:	Pesticide and Herbicide Waste		
	Petroleum distillates	241	300
Class 13:	PCB Wastes		
	PCB	242	80
Class 14:	Clean-up Residuals		
	Toluene	521	1400
	Petroleum distillates	531	1400
	TOTAL		59 410

Source: OWMC (1984)

where Q = emission rate for species i, g/s

K_g = the gas mass transfer coefficient for species i, m/s

A = area of the spill, m^2

P_i = vapor pressure for species i, Pa

M_i = molecular weight of species i, g/mol

R = gas constant, $(Pa.m^3)/(mol.K)$

T = temperature, K

The gas phase mass transfer coefficient was estimated by a method developed by Mackay and Matsugu (1973) which is explained in detail in Appendix C of this report.

$$K_g = 0.0048\ (U)^{0.78}\ (d)^{-0.11}\ (Sc)^{-0.67}$$

where U = wind speed, m/s

d = diameter of pool, m

Sc = Schmidt number, dimensionless

REAGENTS AND ADDITIVES

The reagents and additives for comparison of relative risks were selected based on the potential for the commodities to cause releases to the air as a result of volatilization of organic chemicals or emissions of particulates during a spill event. The reagents and additives used in the physical/chemical treatment and incineration processes are shown on Table E-2. Those reagents and additives determined to be potentially significant contributors to air emissions in the event of a spill were cement, hydrated lime, activated carbon, and calcium oxide. Caustic soda was not selected

TABLE E-2

REAGENTS AND ADDITIVES

Chemical/Reagent	Form	Quantity (t/a)[1]
Physical Chemical Treatment Plant		
Cement	powder	52 700 for total solidification
Sodium silicate	liquid	21 700
Caustic soda	50% liquid	640
Hydrated lime	solid	7400
Activated carbon	solid	100
Nitrogen[2]	liquid	8000 m^3/a
Incinerator System		
Caustic soda	50% liquid	5600
Calcium oxide	solid	2380
Nitrogen[2]	liquid	16 000 m^3/a

[1] All quantities based on information contained in Phase 4 Engineering Report prepared by Monenco (1985).

[2] Quantities at standard temperature and pressure.

because it is usually shipped in flakes or beads when in solid form and would not readily be subject to wind dispersal (Environment Canada, 1981). The other reagents and additives which were excluded are liquids that are not expected to contain volatile organic chemicals.

In the event a spill of reagents or additives occurs, solid materials would most likely form a waste pile. The waste pile would be subject to erosion and wind dispersal of particulates. The estimates of release potential were determined based on the following equation for estimating particulate emissions from a waste pile (EPA, 1977):

$$M_v = 0.0045(M_{wp})(X_i)/(PE)^2$$

where M_v = constituent particulate emission rate, kg/d

M_{wp} = mass of pile, kg

X_i = mass fraction of constituent, kg/kg

PE = Thornthwaite's precipitation-evaporation index

A value of 110 was used for PE which reflects climatic conditions in the Niagara region of the Great Lakes (EPA, 1977). In addition, the spill event was assumed to be a spill of 100% of the commodity being transported.

In the absence of LD_{50} values for the chemicals and additives selected for evaluation, a derived LD_{50} was approximated on the basis of the Threshold Limit Value (TLV). The TLV is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek, to which nearly all workers may be repeatedly exposed without adverse effect. The concentration based TLV was first converted to an equivalent dose based on the following equation:

$$\underset{(\mathrm{mg/kg})}{\text{Equivalent Dose}} = \frac{\mathrm{TLV} \times \mathrm{BR} \times t \times \mathrm{CF}}{\mathrm{BW}}$$

where TLV = Threshold Limit Value, mg/m^3

BR = Breathing rate, 29 l/min

t = Exposure time, 60 min

BW = Body weight, 70 kg

CF = Conversion factor

A conversion factor must be applied to consider the fact that the TLV is based on exposure for an 8-hour workday and a 40-hour workweek and the LD_{50} considers a one hour exposure time. A factor of 1000 was then applied to the LD_{50} equivalent based on the relationship between the TLV and the LD_{50} (McNamara, 1976) assuming that the TLV is equivalent to the no effect level (NOEL).

The quantity of wastes used in the calculation of the hazard index are shown on Table E-2. These quantities were based on the ultimate plant throughput of 300 000 t/a. The quantity of cement required for total solidification was chosen as representing the most conservative case, i.e., total solidification, in determining the severity index.

INTER-SITE TRANSFERS

The relative risks of inter-site transfers were assessed based on those waste streams which could potentially be transported between treatment units located on different sites. Those waste streams identified included oily wastes after treatment by emulsion breaking and dissolved air flotation at the physical/chemical treatment plant and transported to the incineration site; and ash from the incineration unit transported to the engineered landfill. Surrogate chemicals were chosen to represent the oily waste streams that would be sent to the treatment plant (see Appendix B). The commodities selected to represent inter-site transfers and their quantities are presented in Table E-3.

The release potential for the surrogate chemicals was estimated using the equation presented for volatilization of organic chemicals. The release potential for the ash was estimated using the equation presented for emissions of particulates for waste piles.

Toxicity for 1,1,1-trichloroethane and trichloroethylene was based on the LD_{50}. An LD_{50} was unavailable for incinerator ash, therefore, an equivalent dose was estimated from the TLV using the methodology presented for reagents and additives.

TABLE E-3

INTER - SITE TRANSFERS

Waste	Quantity (t/a)
Oily Wastes Trichloroethylene Benzene	8000
Ash	13 000[1]
TOTAL	23 600

[1] Quantity based on information contained in Phase 4A Engineering Report (Monenco, 1985)

E.3 RESULTS AND ANALYSIS

The results of the hazard index are shown below:

Commodity	Overall Severity Index	Frequency Index (t/a)
Special Wastes	7.43×10^{-2}	59 410
Reagents and Additives	8.54×10^{-8}	62 580
Inter-site Transfers	2.06×10^{-6}	21 000

It should be noted that the indices presented above are relative measures of severity and frequency only and do not have any absolute significance. The indices show that the relative risks of the severity of special wastes transported to the facility, reagents and additives transported to the facility, and inter-site transfers are on the order of of 36 000:0.04:1. In addition, the ratio of relative frequency of spills for the three commodity groups is determined to be on the order of 2.8:3:1. Although a spill of reagents and additives can occur at approximately the same frequency as a spill of special wastes, the relative severity of the risks posed by reagents and additives is much less significant in comparison to special wastes. Thus, it can be concluded that the risks associated with transportation of special wastes to the facility are much greater than for the transportation of other commodities.

Since these risks were based on average weather conditions in Southern Ontario, consideration was given to how different conditions might influence the relative risks. In particular, lower temperatures and higher wind speeds would tend to decrease the emission rates for volatile organic chemicals (eg., special wastes) relative to particulates (eg., reagents and additives). For example, the emission rate for a volatile organic chemical at 0°C will be approximately 1.8 times lower than at 10°C. In addition, wind speed influences the emission rate for volatiles by its effect on the mass transfer coefficient, Kg. Higher wind speeds will increase the mass transfer coefficient and thus the emission rate. If the wind speed were changed from 3 m/s to 1 m/s, the emission rate would be reduced by a factor of 2.4. The combined effect of a 10°C decrease in temperature and reduction in wind speed from 3 m/s to 1 m/s would be to reduce the overall severity index for special wastes to 1.77×10^{-2}. It is thus apparent that changes in temperature and wind speed will not have a significant effect on the ratio of relative risks.

REFERENCES

Canviro Consultants, Ltd. 1985. Market Assessment Program: Survey of 1000 Industrial Establishments.. Prepared for Ontario Waste Management Corporation.

Environment Canada. 1981. Technical Information for Problem Spills - Sodium Hydroxide. December.

Franklin Associates, Ltd. 1983. Revised Draft Report on Waste Oil Management and Composition. USEPA Office of Solid Waste.

Mackay D., and Matsugu, R.S. 1973. Evaporation Rates of Liquid Hydrocarbon Spills on Land and Water. Canadian Journal of Chemical Engineering. 51:434, August.

McNamara, B.P. 1976. Concepts in Health Evaluation of Commercial and Industrial Chemicals. In Advances in Modern Toxicology, Volume 1, Part 1: New Concepts in Safety Evaluation. Hemisphere Publishing Co. Washington.

Monenco. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Engineering. Prepared for Ontario Waste Management.

Ontario Waste Management Corporation. 1982. Facilities Development Process. Phase 1 Report. Prepared for Ontario Waste Management Corporation. September

Ontario Waste Management Corporation. 1984. Waste Profile. November.

Stiver, W. and Mackay, D. 1982. Spill Technology Newsletter, January/February 1982. As cited in Technical Information for Problem Spills. Draft Introduction Manual.

United States Environmental Protection Agency. 1977. Compilation of Air Pollutant Emission Factors. Third Edition, August.